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# Pulmonary Sarcoidosis*

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The purpose of this communication is to emphasize the importance of recognizing sarcoidosis as a disease entity particularly by its manifestations in the lungs as observed in the roentgenogram of the chest. In recent years chest radiography was increased to large proportions through greater interest in the value of radiologic findings in general, through tuberculosis surveys of civilian populations and by its routine use in the examination of personnel in industry and in the armed services. Many cases of pulmonary changes in sarcoidosis have been discovered by roentgen examination alone, either by routine or selective studies, and a smaller proportion have had their diagnosis confirmed either by biopsy of a peripheral lesion or by autopsy. Since sarcoidosis is no longer thought of as a rare disease[15], its diagnosis should be readily suspected when the characteristic lesions are present in the radiograph of the chest.

Sarcoidosis, as it is known at the present time, is a generalized disease affecting almost every organ of the body. It has been variously termed lupus pernio of Besnier, multiple benign skin sarcoids of Boeck, Hutchinson's disease, Schaumann's disease or lymphogranuloma benigna, ostitis tuberculosa multiplex cystica (Jungling) and uveo-parotid fever (Heerfordt). According to Hunter[7], the disease was first recognized clinically by Hutchinson as early as 1869 and later described by Besnier[2] as *lupus pernio* in 1889. Boeck[3] was the first to describe the histological features of the skin manifestations and introduced the name *sarcoid* because of the sarcoma-like appearance of the epithelioid connective tissue cells in the perivascular lymph spaces and the occasional giant-cell which was considered to be of sarcomatous type. While Kienbock[10] (1902) Kreibich[13] (1904) and Reider[20] (1910) recognized the radiologic appearance of the cystic changes in the small bones of the hands and feet, it was not until 1917 that Schaumann[21] identified lupus pernio and Boeck's sarcoid as the same disease by the microscopic appearance of the lymph nodes and the bone lesions. Again in 1919, Jungling[9] proved the identity of the cystic lesions observed radiographically by biopsy of the soft tissues of a finger, found the characteristic microscopic changes described by Boeck and applied the term *ostitis tuberculosa multiplex cystica* to these bony changes. In 1915 Kutznitsky and Bittorf[14] reported a case of skin sarcoid with pathologic changes occurring in the spleen and lymph nodes and radiologic findings of mottled infiltration in the lungs and enlargement of the hilum nodes. Schaumann[22] (1924) later gave the name of lymphogranuloma benigna to this disease and while it simulated lymphogranuloma maligna (Hodgkin's disease) in its protean manifestions it tended to follow a benign course. *Uveoparotid fever* described by Heerfordt[6] in 1909 was later recognized as another manifestation of Besnier-Boeck's disease by Bruins-Slot[4] in 1936, and 1937 Longcope and Pierson[16] also attributed the uveal tract and parotid involvement to sarcoidosis.

A series of 19 cases of sarcoidosis have been observed on various services at City Hospital and the University Hospitals of Cleveland of Western Reserve School of Medicine in the past decade, 15 of which were proved by biopsy of a peripheral lesion and 4 by autopsy. A number of other cases were also observed in which the diagnosis was made clinically and roentgenologically, but because of lack of histologic proof they are not included in this report. The most striking feature of the 19 cases was the pulmonary

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and mediastinal lymph node involvement as observed on the chest roentgenogram. Table I includes all the positive findings in the whole series of cases and Table II gives the distribution of the lesions according to anatomical site.

The average age of the series was 29.9 years, the youngest being nine and the oldest sixty-three. There were twelve females and seven males and twelve patients were negro and seven were white. The predominance of negro patients is in accord with the findings of Longcope[15] and Thomas[23].

## ETIOLOGY

The etiology of sarcoidosis is not known, most writers on the subject offering no explanation as to causative factors. Pinner[17] holds to the belief that the disease is a form of non-caseating tuberculosis and supports this view mainly on the basis that some of the reported cases have developed a rapidly progressive form of tuberculosis.

The fact that tubercle bacilli cannot be demonstrated in the sections of tissue or following injection of some of the macerated tissues into animals, and that the tuberculin test is negative in the majority of cases has led most students of the subject to discredit tuberculosis as a causative agent. Kissmeyer and Nielson[12] believe, in view of the repeated negative bacteriologic studies, that the disease is due to an unidentified virus. Rabello[10] finds the lesions of cutaneous and osseous sarcoid to resemble leprosy. The latter concept seems unlikely in view of the striking difference of the radiographic appearance of lesions affecting the phalanges in the two diseases.

## PATHOLOGY

The lack of caseation and the absence of tubercle bacilli in the histologic section are the two main distinguishing features between the tubercle of sarcoid and that of tuberculosis. The sarcoid tubercle is composed of pale-staining polygonal epithelioid cells with an occasional giant-cell usually of the foreign body type (Figures 7A and 7B). Central necrosis is rarely seen and a peripheral inflammatory reaction is usually absent. In the occasional case there may be a slight degree of central necrosis and a few scattered lymphocytes in the peripheral zone. Giant cells are not always present. Healing may take place by fibrosis and hyalinization. Grossly the lesions are firm, gray "hard" nodules from a few millimeters to about one centimeter in diameter depending upon the extent and the degree of coalescence.

## CLINICAL AND ROENTGENOLOGICAL FEATURES

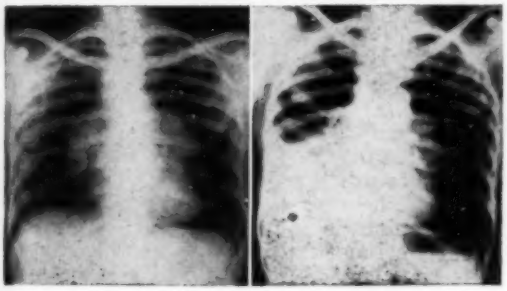
The paucity of clinical signs and symptoms in sarcoidosis is usually the rule. There may be varying degrees of chest findings including cough, expectoration, dyspnea and rales. Examination of the sputum and gastric contents fail to reveal the presence of tubercle bacilli. Fatigue, weight loss and occasionally a low grade fever may be manifestations of the generalized disease. Bizarre phenomena may result from involvement of vital organs such as the case of diabetes insipidus reported by Tillgren[24] in which a focus of sarcoid was found in the pituitary gland. Multiple lesions may occur in the myocardium with changes in cardiac rhythm and evidence of congestive failure[5,1]. External manifestations may present themselves by enlargements of the peripheral lymph nodes, by skin changes of either the Besnier-Boeck or Darier-Roussy types[5], by involvement of the iris, ciliary body, lacrimal, parotid and submaxillary salivary glands, and by enlargement of the spleen, liver or various portions of the skeletal system. The blood cell count is usually within normal limits although polycythemia has been reported. The large monocytes may be increased in number during the active phases of the disease. Eosinophilia is occasionally encountered. Serum globulin is almost always elevated showing a reversal of the A-G ratio. Tuberculin anergy is found in about 70 per cent of the cases. The tuberculin test was positive in four of the present series of 19 cases.

All of the 19 cases reported in this series showed roentgenological changes in the chest consisting of enlargement of the tracheobronchial lymph nodes and various forms of pulmonary infiltrations[11]. (Figures 1A and B; Figures 2A and B). The diffuse small nodular and streaked shadows of increased density radiating outwards from the hila are the changes encountered most consistently. Occasionally the closely packed miliary type of nodulations interspersed by thin strand-like infiltration are observed (Figures 4 and 6). The phalanges of the hands and feet were involved in only 1 case in this series (Figures 5A and B). Cystic defects are found in medullary portions of the phalanges without causing destruction of the cortex and without any evidence of surrounding inflammatory changes. Other portions of the skeletal system may be involved by destructive lesions (Figures 3B, C, and D).

Some of the features of sarcoidosis may best be considered by a review of several cases included in this series.

*Case No.* 2: N. M., a colored female, aged forty-five, was admitted to the hospital on September 26, 1938, complaining of weakness of one year's duration and pain in the left chest for three weeks. Examination revealed rales in both lung bases and the spleen was slightly enlarged. Chest radiography revealed bilateral hilum node enlargement and a slight degree of streaked infiltration in the right base (Figure 1A). A tentative diagnosis of Hodgkin's disease was made. Tuber-

culin tests down to 1:100 dilutions were negative. Repeated sputum examination including concentration technic were negative for tubercle bacilli. A moderate anemia was present. High voltage roentgen irradiation was administered to the hilum nodes resulting in slight diminution in their size. Biopsy of an axillary node in November and repeated in December was reported as chronic hyperplasia and tuberculosis respectively. An excised skin lesion was reported as pityriasis rosea. Additional tuberculin tests were performed with P. P. D. and again proved negative. The liver and spleen became palpable a few cm. below the costal margins. The patient was discharged on January 11, 1939, in fair condition but with a poor prognosis and was followed in the Chest Dispensary. She was readmitted on September 15, 1939, with a pleural effusion in the right base, aspiration of which revealed a serous effusion (Figure 1B). Cultures were negative for py-



(Figure 1A) (Figure 1B)

Fig. 1A. Case No. 2. Sept. 27, 1938. Tremendously enlarged hilum nodes, bilaterally. Right paratracheal nodes moderately enlarged. Thickening of interlobar septum on right.

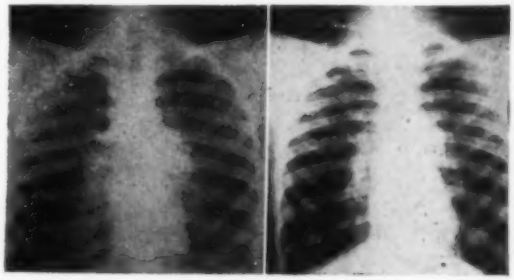
Fig. 1B. Case No. 2. Sept. 18, 1939. Hilum nodes moderately enlarged. Streaked infiltration in right lower lung with large right pleural effuffsion. Autopsy revealed sarcoidosis.

ogenic and tuberculous organisms. Tuberculin tests were again repeated with P. P. D. and a delayed atypical positve reaction was obtained to a double dose of second strength P. P. D. A persistent microscopic pyuria was noted and urinary sediment was negative for tubercle bacilli. The patient gradually became weaker and died on December 26, 1939. Autopsy (Dr. H. H. Macumber) revealed sarcoidosis of the cervical hilum, axillary, portal and superior pancreatic lymph nodes, lungs, liver, spleen and adrenal glands. The spleen weighed 575 grams. Guinea pig inoculation with material from a hilum lymph node yielded negative results.

*Comment*: The diagnosis of sarcoidosis was not made during life. The clinical picture, the lymphadenopathy, the typical histologic picture of the involved organs, the absence of tubercle bacilli and the anergic reaction to high concentrations of tuberculin are all characteristic features of sarcoidosis. The effusion in the right pleural cavity was an unusual finding.

*Case No.* 3: F. A., a 22 year old white male, was admitted to the hospital on July 5, 1939, with marked swelling in both parotid regions and of his eyelids which had first appeared five weeks previously. Examination revealed hard, almost immobile enlargement of both parotid, submaxillary and lacrimal glands. The temperature was 37.2 degrees C. The clinical impression was that of Mickulicz disease. Tuberculin tests were negative with O. T. down to 1:100 dilution and with second strength P. P. D.

A radiograph of the chest on July 13, 1939, revealed "potato-like" enlargements of both groups of hilum nodes (Figure 2A). A biop-



(Figure 2A) (Figure 2B)

Fig. 2A. Case No. 3. July 15, 1939. Greatly enlarged hilum and paratracheal lymph nodes. Biopsy of parotid gland revealed sarcoidosis.

Fig. 2B. Case No. 3. June 6, 1940. Hilum and paratracheal nodes smaller. Diffusely scattered nodular and streaked pulmonary infiltration.

sy from the left parotid gland revealed hyperplastic tuberculosis but was later considered to be Boeck's sarcoid. Special stains failed to reveal the presence of tubercle bacilli. The patient was discharged from the hospital on July 25, 1939, without having received any active therapy and the prognosis was considered good. A follow-up chest radiograph made on June 12, 1940, showed moderate diminution in the size of the hilum nodes and a diffuse nodular and streaked infiltration throughout both lungs (Figure 2B).

*Comment*: This case had the clinical features of Mickulicz disease and uveo-parotid fever although there were no eye changes and the temperature was normal. The radiographic features in the hilum nodes and lungs were characteristic of sarcoidosis.

The biopsy from the parotid gland showed microscopic features consistent with a diagnosis of Boeck's sarcoid (Dr. H. Z. Lund).

*Case No.* 4: B. P., a white female, aged 9, was admitted to the hospital on July 5, 1939, with a history of loss of strength and appetite since August, 1938, and a stiff neck beginning October, 1938. Radiographs of the cervical spine and chest showed destructive

(Figure 3A) (Figure 3B) (Figure 3C) (Figure 3D)

Figure 3A. Case No. 4. July 12, 1939. Slight pulmonary infiltration radiating outwards from left hilum. Destruction of anterior right fourth and fifth ribs and posterior left sixth rib.

Fig. 3B. Case No. 4. Dec. 14, 1939. More advanced destruction of anterior right fourth and fifth (biopsy) and posterior left sixth rib due to sarcoid infiltration.

Fig. 3C. Case No. 4. Feb. 10, 1941. Mottled appearance of second to sixtxh cervical vertebrae due to destruction and new bone production by sarcoidosis (Biopsy).

Fig. 3D. Case No. 4. June 5, 1941. Destruction of pubic bones at symphysis considered to be part of generalized sarcoidosis.

changes from C-1 to C-6 inclusive and destruction in the anterior ends of the right fourth and fifth ribs, (Figures 3B and C), with scattered nodular and streaked infiltration radiating outwards from both hila (Figure 3A). Tuberculin tests down to 1:100 dilution and second strength P. P. D. were negative. For a period of six months there was a gradual increase in visual disturbance with blurring and also the development of some deafness. Diagnoses of primary optic atrophy and chronic serous otitis media with secondary deafness were made. Biopsies were made of the spinous process of the third cervical vertebra, of the posterior portion of the left sixth rib, which were reported as chronic inflammation. Later the patient developed swelling of both sides of the mandible and had difficulty in opening her mouth. Biopsy made of the left mandible was reported as chronic destructive inflammation of bone, non-suppurative.

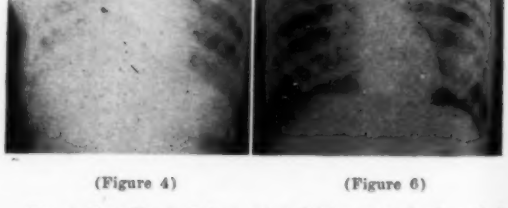
Radiograph of the pelvis on June 5, 1941, showed irregular destruction of the symphysis pubis (Figure 3D).

All the biopsy material was sent to Dr. Henry L. Jaffe, pathologist for the Hospital for Joint Diseases of New York City, who believed the diagnosis was sarcoid. Additional tuberculin tests were made all of which proved negative. The patient was discharged on June 26, 1941, to the State Institute for the Blind.

*Comment*: This case presents unusual features in that multiple bones are involved in a similar manner. The repeatedly negative tuberculin tests, the infiltration in lungs, and the opinion of an experienced pathologist in bone diseases augur well for a diagnosis of sarcoidosis. It is not certain that the primary optic atrophy can be accounted for by involvement by sarcoidosis, but it could be a possibility since it is known that nerve tissue may be involved[15].

*Case No.* 5: O. S., a colored female, graduate nurse, aged 27, was admitted to the tuberculosis division on July 12, 1940. She gave a history of malaise and mild fatigue of six months duration, followed during the two months prior to admission by progressive dyspnea of such degree that she was conscious of labored respirations when walking. There was also present a hacking cough, usually unproductive, a loss of eight pounds in the six months before admission. She continued to work until three days before entry into the hospital.

Examination revealed a well developed young colored female, dyspneic on exertion but breathing quietly at rest and not appearing seriously ill. The temperature was normal, the pulse rate varied between 80 and 100, and the blood pressure was 112/72. Fine rales were audible in the lower two-



(Figure 4) (Figure 6)

Fig. 4. Case No. 5. July 3, 1940. Diffuse and closely packed nodulation interspersed by streaky infiltration in both lungs. Emphysematous bullae in both apices. Autopsy revealed sarcoidosis.

Fig. 6. Case No. 13. April 21, 1943. Fine miliary nodulation throughout both lungs interspersed by streaked infiltration and slight coalescence in left upper lobe. Autopsy revealed sarcoidosis.

thirds of the right lung and a few scattered rales were present on the left side. The heart, abdomen, liver, spleen and peripheral lymph nodes were negative to examination.

The vital capacity on admission was 1100

cc. or 35 per cent. Hemoglobin, blood cell count and urine were within limits of normal. Tuberculin tests with P. P. D. were negative using first and second strengths, but a positive test was obtained using a double dose of second strength material. The sputum was mucoid in character and repeated examinations of sputum and gastric contents for tubercle bacilli and fungi were negative.

The chest radiograph revealed a symmetrical diffuse nodulation and streaked infiltration of both lung fields sparing the apices, and showed enlargement of the mediastinal lymph nodes (Figure 4).

During her hospital stay the patient developed progressive dyspnea and her paroxysmal unproductive cough became worse. She was afebrile except late in the course of her disease, but the temperature did not exceed 38 degree C. The urine later contained 1+ albumin and the total white blood cell count rose to 18,000. Late in the course of her disease she developed two small lymph node enlargements in the left axilla and a small node in the right axilla. She became progressively worse and despite symptomatic treatment and the administration of potassium iodide died on September 16, 1940, of respiratory and cardiac failure.

(Figure 5A) (Figure 5B)

Fig. 5A. Case No. 6. July 3, 1941. Ovoid punched-out areas of destruction involving the phalanges of the hands. Biopsies from skin of nose, arm and back showed Boeck's sarcoid.

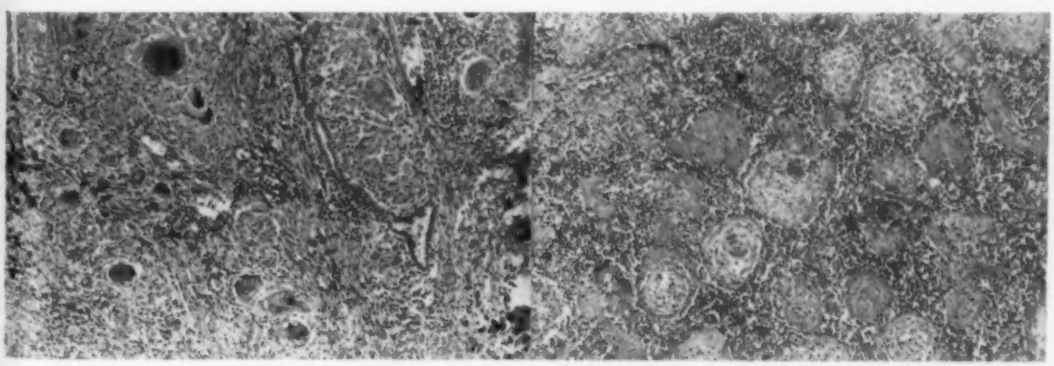
Fig. 5B. Case No. 6. July 3, 1941. Punched-out areas of bone destruction of the phalanges of the feet. Biopsies from skin of nose, arm and back showed Boeck's sarcoid.

The autopsy (Dr. A. E. Margulis) revealed Boeck's sarcoid of the lungs, bronchi, pleura, pericardium, spleen, right kidney, and mediastinal, left inferior deep cervical, pretracheal, celiac, hepatic and para-aortic lymph nodes. The right side of the heart was moderately dilated and slightly hypertrophied (cor pulmonale).

*Comment*: The radiographic appearance of lungs was not unlike that of miliary tuberculosis (Figure 4) with nodular and streaked shadows closely packed except for emphysematous bullae in the apices. With the temperature approximately normal, the absence of tubercle bacilli in the sputum and gastric contents and the tuberculin tests negative in higher dilutions, a diagnosis of tuberculosis seemed unlikely and the final clinical diagnosis was thought to be metastatic neoplasm to the lungs.

*Case No.* 13: R. J., a colored female, aged 15 years, was admitted to the tuberculosis division of City Hospital on April 17, 1943. Her illness began in June, 1942, with cough productive of small amounts of mucopurulent sputum, weakness and gradual weight loss. Two months later she reported to Children's Health Dispensary and was told she had mumps. She continued to be moderately ill until two weeks before admission when she developed fever, night sweats and moderate dyspnea. Routine fluoroscopy in school revealed a pulmonary lesion. Her total weight loss was 37 pounds.

Examination revealed a poorly nourished, chronically ill young girl in no apparent dis-



(Figure 7A) (Figure 7B)

Fig. 7A. Case No. 13. Photomicrograph (x 160) of section of lung showing epithelioid tubercles and considerable interstitial fibrosis. A few scattered giant-cells are present. No caseation or central necrosis.

Fig. 7B. Case No. 13. Photomicrograph (x 142) of a lymph node showing tubercles formed by pale-staining epithelioid cells. Caseation and necrosis are not present.

## TABLE I — PULMONARY SARCOIDOSIS — TABULATION OF FINDINGS IN 19 CASES

| Age | Case No. Initials Sex Color | Date of Admission | Radiographic Appearance of Lungs | Lymph, Nodes, Radiographic and Clinical | Skin | Other Organs | Globulin mgm. Per Cent | Tuberculin Reaction | Remarks |
|---|---|---|---|---|---|---|---|---|---|
| 22 | 1<br>B. M.<br>F W | 1-20-36 | Slight infiltration adjacent to hilum | Hilum bilat. | Evelid*<br>Leg* | | | Neg. | |
| 44 | 2<br>N. M.<br>F B | 9-26-38 | Streaked infiltration right middle and lower lobes | Cervical, hilum, axillary, portal, sup. pancreatic | | Spleen, Liver, Adrenals right pleural effusion | | Neg. | Autopsy<br>(see case report) |
| 22 | 3<br>F. A.<br>M W | 7-5-39 | Disseminated nodular and streaked infiltration | Hilum bilat. | | Parotid glands* | | Neg. | See case report |
| 9 | 4<br>B. P.<br>F W | 7-5-39 | Scattered nodular and streaked infiltration | | | Ribs* C-3*<br>left mandible*<br>Symphysis<br>Pubis—Liver | 4.2 | Neg. | Referred to State Institute for the Blind.<br>Primary optic atrophy |
| 27 | 5<br>O. S.<br>F B | 7-12-40 | Miliary and streaked infiltration | Mediastinal left inferior deep cervical, pretracheal, celiac, hepatic and para-ortic | | Pleura<br>pericardium<br>right kidney | 6.0 | Neg. | Autopsy<br>(see case report)<br>Tuberculin positive double dose second strength P.P.D. |
| 40 | 6<br>F. W.<br>F B | 6-28-41 | Slight nodular infiltration | Hilum bilat. | Nose*<br>Arm*<br>Back* | Phalanges of fingers and toes bilat. | 4.25 | Neg. | Tonsil biopsy showed endothelial hyperplasia |
| 63 | 7<br>A. G.<br>F B | 10-4-41 | Streaked infiltration right upper and left lower lobes | | Right foot* | | 6.1 | Pos. | Tuberculin P.P.D. second strength |
| 32 | 8<br>H. O.<br>M W | 10-4-41 | Nodular and patchy infiltration | Hilum bilat.<br>right paratracheal | Back*<br>Left knee | Iris | | Neg. | Many skin nodules on back |
| 23 | 9<br>C. L.<br>F W | 3-20-42 | Slight nodular infiltration | Left inguinal*<br>Generalized superficial<br>right paratracheal<br>Hilum bilat. | | | 4.16 | | Secondary anemia |
| 56 | 10<br>E. M.<br>M B | 7-17-42 | Nodular and streaky infiltration | Left axillary*<br>right paratracheal<br>Generalized superficial | | | 4.5 | Neg. | |
| 21 | 11<br>S. G.<br>M W | 8-11-42 | Streaked infiltration left lower lobe | Cervical*<br>Paratracheal and hilum bilat. | | | 4.2 | Pos. | Tuberculin P.P.D. second strength<br>Nodes disappeared |
| 36 | 12<br>M. H.<br>F B | 12-9-42 | Slight infiltration at left hilum | Right supraclavicular*<br>Hilum bilat.<br>Right paratracheal | | Liver | 3.16 | Pos. | |
| 15 | 13<br>R. J.<br>F B | 4-17-43 | Diffuse miliary and streaked infiltration | Cervical<br>Bronchopulmonary | | Liver, spleen, kidney, colon, submax. salivary gland, lingual and faucial tonsils | 6.0 | Pos. | Autopsy<br>(see case report)<br>Tuberculin P.P.D. second strength |
| 13 | 14<br>J. B.<br>F B | 5-27-43 | Diffuse nodular infiltration | Cervical*<br>Right paratracheal | | Iris, lacrimal gland, parotid liver, spleen | 3.8 | Neg. | All lesions cleared 4-15-46 |
| 55 | 15<br>W. P.<br>M B | 1-28-44 | Streaked infiltration right upper and lower lobes | Inguinal*<br>Left Epitrochlear* | | | | Neg. | |
| 23 | 16<br>G. A.<br>F B | 3-15-44 | Nodular and streaked infiltration | Hilum and paratracheal | Abdomen* | | 6.15 | Neg. | Irreg. fever rising to 39.5 degrees C. |
| 42 | 17<br>E. Y.<br>F B | 4-18-44 | Bilateral infiltration | Mediastinal lymph nodes | | Liver | | | Autopsy<br>Died two days after admission<br>Bronchopneumonia |
| 16 | 18<br>W. S.<br>M B | 11-19-44 | Miliary and streaked infiltration<br>(Reticulated) | Right inguinal*<br>Generalized superficial<br>Hilum and paratracheal | | Uveitis bilat.<br>iris, right | 3.35 | Neg. | Blood smear showed 30 large monocytes |
| 10 | 19<br>C. C.<br>M B | 10-16-45 | Diffuse streaked infiltration | Bilat. hilum<br>right paratracheal | Chest*<br>Neck* | Uveal tract<br>parotid | | Neg. | |

*Biopsy

tress. Abnormal findings were limited to the chest where rales were heard over both lungs. The temperature was 37 degrees C., pulse 90, respirations 22, blood pressure 115/90 and vital capacity 1100 cc. or 33 per cent.

Laboratory studies showed the urine to be normal. Sedimentation rate was 1.33 with hematocritic 41. Blood urea nitrogen was 12.6, total proteins 10.2 grams, albumin 4.2 and globulin 6.0 grams per 100 cc., the A-G ratio being 0.69. Eight sputum concentrations and three gastric washings were negative for tubercle bacilli. Several tuberculin tests were negative, but further testing with second strength P. P. D. was strongly positive. Sputum cultures for fungi and blood cultures were negative.

Roentgen examination of the chest revealed a diffuse miliary nodulation throughout both lung fields with a tendency towards coalescence in the left upper lobe. A fine streaked infiltration was interspersed between the nodules. The hilum lymph nodes were moderately enlarged. No change took place in the appearance of the lungs and mediastinum during an eight months period (Figure 6).

Throughout the eleven months in the hospital, the patient's fever was low grade in character. She gained a few pounds in weight and had no specific complaints other than cough productive of 50 grams of sputum. During the last several months she had a marked tachycardia and increasing dyspnea. On February 26, 1944, a diastolic gallop rhythm was heard over the apex of the heart. During her last week she developed orthopnea, ascites, hepato and splenomegaly with a total weight gain of 14 pounds. Death occurred on March 6, 1944, the 328th hospital day.

The autopsy (Dr. H. Z. Lund) revealed widespread pulmonary fibrosis, both nodular and reticular consistent with a diagnosis of sarcoidosis (Figure 7A). Characteristic lesions of sarcoidosis were found in the liver, salivary glands, colon, kidneys, spleen, cervical and bronchopulmonary lymph nodes (Figure 7B) and lingual and faucial tonsils. Other findings were relative right ventricular hypertrophy, marked pulmonary emphysema, chronic bronchitis, bronchiectasis, fibrous pleural adhesions and right pleural effusion of 500 cc. A portion of the spleen was macerated, suspended in saline solution and injected into the subcutaneous tissues of a guinea pig. The pig died in three weeks without evidence of tuberculosis or other grossly demonstrable disease.

*Comment*: The clinical diagnosis was considered to be far advanced pulmonary tuberculosis. Because of experience gained from Case No. 5 and because of lack of change in the character of the lesions over a period of about eight months, the roentgenologic interpretation late in the course of the disease was pulmonary sarcoidosis. In all probability, the earlier diagnosis of "mumps" was in truth sarcoidosis of the parotid glands (uveoparotid fever).

## DIFFERENTIAL DIAGNOSIS

The diagnosis of sarcoidosis of the lungs and mediastinum is more often confused with pulmonary tuberculosis than with other diseases. The character of the nodulation and streaked infiltration with hilum and paratracheal node enlargements, the lack of fever, the anergic reaction to tuberculin, the elevated serum globulin fraction with reversal of the A-G ratio are distinguishing clinical features between sarcoidosis and tuberculosis. Hodgkin's disease or some other form of lymphoblastoma may simulate sarcoidosis because of widespread lymph node involvement and a biopsy of the peripheral lesion may be required to determine the correct diagnosis. The fine nodulation in the lung fields is rarely found in Hodgkin's disease and is often observed in sarcoidosis. The history, clinical and roentgenologic features of sarcoidosis usually serve well in differentiation from hilum node tuberculosis, miliary carcinomatosis, erythema nodosum, fungus disease, pulmonary hemosiderosis and silicosis. The absolute diagnosis of sarcoidosis is dependent upon histologic studies of the involved tissues.

## TREATMENT

Little can be said regarding specific treatment of sarcoidosis since most of the cases recover spontaneously. Bed rest, arsenic, cod-liver oil, leprosol, gold salts, hyperpyrexia and ultraviolet light have been advocated at one time or another. Radiotherapy, in the hands of some workers [18 1], has produced rapid retrogression of some of the lesions. Further experience will be required before the value of roentgen therapy can be established.

## SUMMARY

The importance of recognizing pulmonary involvement in sarcoidosis is emphasized. Sarcoidosis is a generalized disease affecting almost every organ and system in the body. In past years various eponyms has been used to designate the disease as it affected different anatomical sites such as skin, bone marrow, lymph nodes, lungs, parotid glands and various structure in and about the eye. Nineteen cases with pulmonary involvement and coexisting lesions have been reviewed. Five case reports are included illustrating clinical, roentgenological and pathological manfestations of the disease. The prognosis is good in most of the cases, three having died of sarcoidosis and one from bronchopneumonia. The etiology, pathology, differential diagnosis and therapy are discussed.

## TABLE II—DISTRIBUTION OF LESIONS

| Location | Number | Biopsy | Autopsy |
|---|---|---|---|
| Lungs | 19 | | 4 |
| Lymph Nodes | | | |
| Superficial | 10 | 7 | 3 |
| Mediastinal | 11 | | 2 |
| Bronchial | 12 | | 2 |
| Abdominal | 2 | | 2 |
| Skin | 6 | 6 | |
| Bones | 2 | 1 | |
| Eyes | | | |
| Iris | 3 | | |
| Uveal tract | 2 | | |
| Lacrimal Gland | 1 | | |
| Salivary Glands | | | |
| Parotid | 3 | 1 | |
| Submaxillary | 1 | | 1 |
| Tonsils | 1 | | 1 |
| Liver | 5 | | 2 |
| Spleen | 2 | | 1 |
| Adrenal Gland | 1 | | 1 |
| Pleura | 1 | | 1 |
| Pericardium | 1 | | 1 |
| Kidney | 2 | | 2 |
| Colon | 1 | | 1 |

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## *Announcement*

The $34,000 prize contest for physicians' art work on the subject of "Courage and Devotion Beyond the Call of Duty" will be judged at the Atlantic City Centennial Session of the A.M.A. at Atlantic City June 9-13, 1947; closing date is May 15, 1947.

Art works on other subjects may also be submitted for the regular cups and medals.

For full information, write Dr. F. H. Redewill, Secretary, American Physicians Art Association, Flood Building, San Francisco, Calif., or to the sponsor, Mead Johnson and Company, Evansville 21, Ind., U.S.A.

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Genius in poverty is never feared, because nature, though liberal in her gifts in one instance, is forgetful in another.—*B. R. Haydon.*

---

Whatever government is not a government of laws is a despotism, let it be called what it may.—*Daniel Webster.*

---

Men must not be poor; idleness is the root of all evil; the world's wide enough, let 'em bustle.—*Farquhar.*

---

Our ideals are our better selves.—*A. Bronson Alcott.*

---

they leave that safety in any hands but their own.—*Daniel Webster.*

---

Ill-humor arises from an inward consciousness of our own want of merit, from a discontent which ever accompanies that envy which foolish vanity engenders.—*Goethe.*

---

Babylon in all its desolation is a sight not so awful as that of the human mind in ruins.—*Scrope Davies.*

---

In that worthiest of all struggles — the struggle for self-mastery and goodness — we are far less patient with ourselves than God is with us.—*Timothy Titcomb.*

---

We get impatient, and there crops out our human weakness.—*Timothy Titcomb.*

---

Every subject's duty is the king's; but every subject's soul is his own.—*Shakespeare.*

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# The Rh Blood Factors*

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In view of the apparent confusion concerning the Rh factors of human blood, it seems timely to attempt a general account of the nature of these factors, the tests used in detecting them, and their significance in clinical and medico-legal applications.

## HISTORICAL

Since some animal bloods contain antigens which are similar to certain of those found in human bloods, Landsteiner and Wiener, in 1937, used blood from the rhesus monkey to inject guinea pigs and rabbits in an attempt to produce antisera with which further studies of the M antigen could be made in human bloods. In the course of their experiments, there was discovered in human blood an antigen unlike the M, N, or P antigens which had been previously known. In a series of tests it was found that this new antigen occurred in about 85 per cent of the bloods of white persons tested and was entirely absent from 15 per cent of the bloods tested[1]. Because of the source of the material used in preparing these antisera, namely, the blood of the rhesus monkey, they named the newly discovered antigen Rh (rhesus).

The clinical importance of the antigen was soon demonstrated by Wiener and Peters[2] who observed three cases of transfusion reactions which could not be attributed to reactions between the ordinary antigens, i.e., A, B, or AB but which, in each case, was related to the presence of the Rh antigen in the donor's corpuscles. Landsteiner and Wiener, therefore, published their original findings in regard to the Rh antigen in 1940 and within the following year, Wiener[3] saw ten additional instances of intragroup transfusion reactions which could be traced to the presence of the Rh antigen.

## NATURE OF THE RH ANTIGENS

*The antigen is not a single entity.* Following the work of Landsteiner and Wiener it seemed apparent that a simple test for detecting the Rh antigen in human blood lay in the use of anti-Rh serum produced in the guinea pig. Further work, however, revealed at least two difficulties. In the first instance it is extremely difficult to produce a potent antiserum in animals for testing. It is also now known that all bloods from infants, regardless of whether they are Rh positive or Rh negative, will be acted upon by guinea pig antiserum[4].

It was observed that human serum from Rh negative individuals who had received multiple transfusions of Rh positive blood contained antibodies against the antigen and it was only natural to try this antiserum for testing unknown bloods. Again, however, difficulties were encountered. It was found that the results obtained with such antisera did not coincide with the results observed by Landsteiner and Wiener in their studies on the distribution of the antigen when guinea pig antiserum was used as test material. Three different distribution figures were obtained when human antisera were used[5]. With some human antisera it was found that 87 per cent of the bloods tested were Rh positive, with another antiserum only 70 per cent were positive and with a third antiserum only 30 per cent were positive.

From this evidence it is concluded that the Rh factor is made up of at least three distinct antigens which may occur alone or in combination with one or both of the other antigens in human bloods. These antigens have been named $Rh_0$, $Rh^1$, and $Rh^{11}$ and combinations of these give rise to seven types of bloods which are Rh positive, namely $Rh_0$, $Rh^1$, $Rh^{11}$, $Rh^1Rh^{11}$, $Rh_1$, $Rh_2$, and $Rh_1Rh_2$. The absence of any Rh antigen gives an Rh negative type.

*Importance of the various antigens.* From the clinical standpoint, only the $Rh_0$ antigen need be considered. Of all persons having some Rh antigen in the blood, 85.9 per cent will have $Rh_0$ alone or in combination with $Rh^1$ or $Rh^{11}$. Only 1.2 per cent will have either $Rh^1$ or $Rh^{11}$ in the absence of $Rh_0$ antigen. By testing for the $Rh_0$ antigen, therefore, practically all Rh positive individuals will be detected.

*Antigenicity of the antigens.* Of the three antigens, $Rh_0$ is apparently most antigenic in the sense that it can stimulate the formation of antibodies when injected. Even with this antigen, multiple transfusions are necessary before a dangerous level of antibodies

*Delivered before the Section on Public Health, Oklahoma State Medical Association Annual Meeting, May 2, 1946.

is reached. It is extremely rare that either Rh[1] or Rh[11] antigen is responsible for transfusion accidents and hence, these two antigens need not be considered in routine clinical studies.

*Inheritance of the antigens.* The Rh antigens are inherited according to a Mendelian pattern and offspring from Rh positive parents will be Rh positive. Naturally, offspring from Rh negative parents will be Rh negative.

Racial distribution of the antigens varies somewhat as shown by investigators[6 7 8]. As previously remarked, about 85 per cent of the white population tested is Rh positive. Chinese, Japanese, and full blooded American Indians are approximately 99 per cent Rh positive while about 91 per cent of the negroes in this country are Rh positive.

## TESTING FOR RH ANTIGENS

The usual test for these antigens consists in a hemagglutination test similar to that used for detecting the A, B, AB, and O groups. The test is not difficult to set up but some skill is needed for correctly interpreting the results.

*Testing serum.* As in all serologic tests, some starting point must be known. In these tests the individual having no known antiserum with which to start may obtain such commercially. Once tests have been started individuals will be found who, by fortuitous circumstances, have anti-Rh antibodies in the serum. These may be bled for a small amount of blood and their serum used for further tests. It is to be remembered, however, that such antisera should be treated to remove the alpha and beta antibodies of groups A and B; otherwise, unless the individual whose blood is being tested belongs to group O, reactions with the A or B antigen will be encountered. These may be absorbed with A and B corpuscles to remove the alpha and beta agglutinins. By following such procedures any hospital laboratory should be able to maintain sufficient stocks of testing sera.

*Corpuscle suspension.* Since the Rh antigen occurs in the red corpuscles, suspensions of these are used for testing. A few drops of blood from the ear or finger may be caught in saline and will be sufficient for testing. As a rule, better results will be obtained if the corpuscles are washed with saline and made into a 2 per cent suspension. Fresh blood will give better results than old suspensions.

*Test proper.* To set up the test, one drop of the corpuscle suspension is mixed with one drop of the testing serum in a small test tube and incubated in a water bath at 37° C. until the corpuscles settle out. This usually requires from 30 to 60 minutes. If a more rapid test is desired the tubes may be lightly centrifuged after a 30 minute incubation period to facilitate agglutination. If the blood is Rh positive agglutination will be evident. Controls of known Rh positive and known Rh negative corpuscle suspensions must be included in the protocol.

While it is not advisable to attempt tests on the open slide because of erratic results, a recent report has indicated that such tests may be set up if antiserum having an anti-Rh titre of 1:1000 or more is available[9].

When known Rh positive bloods are available, tests may be run on sera for anti-Rh antibodies. This test is set up much the same as the one above except the patient's serum is made up in serial dilutions and tested against the known Rh positive corpuscle suspension for agglutinins.

It may be mentioned here that a supplementary test for compatibility of bloods as regards the Rh antigens has been used by Wiener[10]. This is a biological test and is not to be used to supplant the regular test for the Rh antigens. Briefly, it consists in drawing 10 cc of the patient's blood, 5 cc of which is allowed to clot and 5 cc of which is citrated. Fifty cubic centimeters of the prospective donor's blood then is injected intravenously into the patient. After one hour, another 10 cc sample of the patient's blood is drawn and treated as before. The citrated samples are then centrifuged at once and the color of the sera in the two tubes is compared. The clots in the clotted samples are separated and the sera are examined for any change in color. If there is any evidence of hemolysis in any of the tubes, this is evidence of the incompatibility of the donor's and recipient's bloods. If no change occurs, the transfusion may be given carefully, watching for any signs of reaction in the patient.

## CLINICAL SIGNIFICANCE

From the clinician's point of view, two conditions in which the Rh antigen is significant present themselves.

Early in the work with the Rh antigen, it was noted that transfusion reactions due to this antigen were seen in two types of cases: 1. Those in which Rh negative patients had received multiple transfusions with Rh positive blood and 2. those in women who showed a reaction on primary transfusion.

*Multiple transfusions.* This situation may be easily clarified. Since the Rh antigens are antigenic and will cause the formation of anti-Rh antibodies during a series of transfusions, multiple transfusions from an Rh positive donor may lead to the immunization or sensitization of an Rh negative patient so that a subsequent transfusion with Rh positive blood produces severe hemolytic reactions. The occurrence of such type reactions is not high, however. There is an apparent difference in the ease with which Rh

negative individuals may be sensitized to these antigens and it is estimated that not more than 2 per cent to 4 per cent of those receiving from 10 to 20 transfusions will be sensitized.

*Iso-immunization and erythroblastosis fetalis.* The second set of reactions were found to occur in whomen who had recently been pregnant, delivering eighter a normal infant or one who had erythroblastosis fetalis. Further study of these cases lead Levine, Katzin, and Burnham[11] to suggest that this situation is due to iso-immunization of an Rh negative woman carrying an Rh positive fetus. In some as yet unexplained manner, antigen from the fetus goes across the placental barrier to cause the formation of anti-Rh antibodies in the maternal circulation and when the antibody titre becomes high enough, the antibodies pass from the mother to the fetus leading to severe reactions in the fetus or even death. In addition to the possible danger to the infant or fetus, the mother, being sensitized to the Rh antigen, is susceptible to a severe reaction if transfused with Rh positive blood.

In these cases, sensitization of the mother or fetus probably occurs in not more than one in 500 pregnancies. The condition usually does not occur in primiparas, probably because the antibody titre does not reach a dangerous level during a single pregnancy. In multiparas, however, the danger is greater because of the cumulative effect of sensitization instituted during a former pregnancy.

*Transfusion in the Rh sensitized individual.* In all the conditions described above, whether an Rh negative individual becomes sensitized through multiple transfusions, through carrying an Rh positive fetus, or in the case of the infant, through the transfer of antibodies from the mother, transfusions should be made with Rh negative blood. To do otherwise may result in a severe or even fatal accident. It is well to remember also that Rh antibodies have been shown to be transferred in breast milk[12] and infants exhibiting any signs of erythroblastosis should not be fed mother's milk.

It has been suggested in following the course of a pregnancy, especially in those in which the fetus may be Rh positive and the mother Rh negative, titrations of the maternal serum should be made at intervals. If the titre of anti-Rh antibodies rises higher than 1:10, preparations should be made to have known Rh negative blood available for transfusions if needed. It has further been suggested that if the titre goes above 1:10 at or about the eighth month of pregnancy, labor may be induced to save the infant from severe erythroblastosis fetalis.

*Erythroblastosis fetalis due to the Hr antigen.* While the conditions described above will account for over 90 per cent of the cases of erythroblastosis fetalis, it may be expedient to interject a few words concerning another cause of this condition.

Levine, Javert, and Katzin[13] called attention to a case which did not follow the usual conditions seen in other cases. They observed a case which occurred in an infant of Rh positive type and whose mother was also of Rh positive type, a situation contrary to that expected. They showed, furthermore, that serum from the mother contained hemagglutinins active against an antigen in all Rh negative bloods and those Rh positive bloods which did not contain the $Rh^1$ antigen. It was apparent that the case was one involving an antigen distinct from the Rh antigen and due to its peculiar distribution, occurring as it did in all Rh negative bloods, they named the new antigen Hr in contra-distinction to Rh.

Race and his co-workers[14] in England also pointed out the occurrence of this antigen and through their work which was more accurate than that of Levine by virtue of the use of a more potent antiserum, it is now concluded that all individuals including those who are Rh negative are Hr positive except those who are of $Rh^1$ or $Rh_1$ type. In view of the rarity of the Rh' type, to all practical intents, only persons who are $Rh_1$ type are Hr negative.

In cases of erythroblastosis fetalis due to the Hr antigen, the mother must necessarily be of $Rh^1$ or $Rh_1$ type, but the fetus may be of any Rh type except $Rh_0$, $Rh_2$, or $Rh^2$.

Just as in cases of erythroblastosis fetalis due to the Rh antigen, a store of known Hr negative blood should be at hand in the event transfusions are needed. The blood must necessarily be of $Rh^1$ or $Rh_1$ Hr negative type.

## MEDICO-LEGAL APPLICATIONS

Little need be said concerning the value of blood grouping tests in medico-legal applications. In general, such tests are applicable in cases of disputed parentage, identification, and criminal cases. While excellent results have been obtained through tests for A, B, M, N, and P antigents, the addition of tests for the Rh antigens further enhances the value of such tests.

*Cases of disputed parentage.* Inasmuch as the Rh antigens are inherited, two general laws can be applied in cases of disputed parentage. These are 1. The antigens $Rh_0$, $Rh^1$ or $Rh^{11}$ cannot appear in the blood of an infant unless present in the blood of one or both parents and 2. A type $Rh_0$ or Rh negative parent cannot have a child of $Rh_1Rh_2$ or $Rh^1Rh^2$ type, neither can a parent

of $Rh_1Rh_2$ or $Rh^1Rh^{11}$ type have a child of $Rh_0$ or Rh negative type.

*Identification tests.* On the basis of the heredity of the Rh antigens, an individual will carry a certain blood group antigen pattern through life, thus it would be impossible for him to completely change his identity. While it is not possible to identify an individual on the basis of his blood group antigen pattern, it is possible to exclude others not having the same pattern. Thus, illegal claimants to estates or infants interchanged inadvertently in the hospital may possibly be detected by the application of the blood group tests including the Rh antigen determinations.

*Exclusion in criminal cases.* The study of blood stains found at the scene of a crime or on the person of the victim will often do much toward excluding certain suspects. If accusations are made and the blood stains can be shown to be of different antigenic make-up from that of the victim and also from that of the accused, there can be no case against the accused.

In all the applications above, however, one must keep in mind that such applications can be used only as exclusion tests and not as positive proof of identity.

## SUMMARY

1. About 85 per cent of the bloods of the white population contain an antigen known as the Rh antigen.

2. While this antigen is composed of at least three antigens in combination or alone, only the $Rh_0$ antigen is of great clinical importance.

3. The Rh antigens are inherited.

4. Blood grouping tests to study the Rh antigen should be done in the following cases:

   a. The individual has received multiple transfusions and thus may have become sensitized to the Rh antigen.

   b. A woman has borne children or is pregnant and is to receive a blood transfusion.

   c. A newborn infant exhibits jaundice or edema. The blood of the mother and father in this case should also be studied in order to exclude or ascertain the possibility of erythroblastosis fetalis.

5. If, in the conditions described above, the individual is found to be Rh negative, only Rh negative blood should be used in transfusions to the individual. This applies as well to the Rh positive infant who is suffering from erythroblastosis fetalis and who requires blood transfusions during the first few weeks of life. Obviously in the latter case, transfusions with his mother's blood is absolutely contraindicated.

6. In cases of early pregnancy or in a woman who is anticipating pregnancy the blood of both the husband and the wife should be examined for the Rh antigen. In the event that the husband is Rh positive and the wife is Rh negative, the possibility of erythroblastosis fetalis must be recognized. The first pregnancy carries little danger to the fetus, but subsequent pregnancies should be closely followed.

7. Periodic examinations for quantative determinations of anti-Rh antibody in the maternal circulation may give some indication for section at about the eight month to protect the infants against erythroblastosis.

8. In the event that Rh testing facilities are not available, remember that a pregnant woman who is possibly sensitized to the Rh antigen and needs a transfusion should not be transfused with the blood of the husband even if it is of the same blood group. In such a case, if the woman has become sensitized through pregnancy, the husband will be Rh positive in 100 per cent of the cases. In the population as a whole, only 85 per cent are Rh positive and the mother would have one chance in seven of receiving Rh negative blood from anyone else than the husband.

9. In a few cases of erythroblastosis fetalis, the explanation lies in the occurrence of a distinct antigen in all Rh negative individuals and in most Rh positive individuals. This antigen is designated as the Hr antigen.

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## DISCUSSION

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Doctor Marsh has given us a splendid discussion of a subject which is important to us all and about which we know too little. It is important because of the serious consequences which can result from a lack of understanding of the factors involved.

Without attempting to add to what has been said I shall only try to emphasize some of the most important points. As was pointed out the Rh factor is not composed of one single substance but has several components. However, in this discussion we shall speak of it as a single item. It is something that is present in the blood of 85 per cent of our population and absent in that of the other 15 per cent. That should cause us no more concern than the fact that a certain per cent of the people have blue eyes and another per cent have brown eyes were it not for the discovery that this substance possesses antigenic properties. In other words if it happens to get into the blood of an individual who does not normally possess it, the formation of protective antibodies may be stimulated and this antigen antibody reaction may be so violent as to produce harmful or even fatal results.

There are two methods by which this Rh factor may get into the circulation of Rh− individuals in such a way as to cause reactions. First of all it may happen by giving repeated transfusions of Rh+ blood to an Rh− individual. Reactions do not by any means always follow for as Doctor Marsh pointed out, even after as many as 15 or 20 such transfusions, reactions will be seen in only from four to eight per cent of the recipients.

The other instance in which such reactions may arise is when an Rh− woman is pregnant with an Rh+ baby. By some means the Rh factor passes the placental barrier into the mother's blood and stimulates the formation of antibodies. These find their way back into the baby's circulation and by their action against his blood, injure or destroy him. As was pointed out such reactions by no means invariably take place and when they do show up it is not apt to be in the first pregnancy but in subsequent ones.

Now why do such reactions not take place when the first Rh+ blood is transfused into an Rh− individual and often not when any number of such transfusions take place? Likewise, why is the firstborn child pretty sure to escape having erythroblastic anemia while those of later pregnancies may have it? The reason is, of course, that it takes time for the antibodies to be formed and even then the formation of protective antibodies may be such an orderly process that no severe reaction takes place. As a matter of fact, the individual has, since the time he first began to ingest food, and to come into contact with various antigenic substances, had to be manufacturing various protective antibodies. This process generally goes on in such a quiet way that no particular disturbance is caused. But a certain per cent of people do this in an abnormal sort of way or, speaking more correctly, they overdo the process, so that there comes to be such a violently antagonistic antigen antibody reaction as to cause disturbing symptoms. Such persons may have what we call allergic reactions. If one of these is Rh− and he gets repeated transfusions with Rh+ blood he may show transfusion reactions. Or if the individual be an Rh− woman and she have repeated pregnancies with Rh+ babies, erythroblastic anemia may show up. The 4 to 8 per cent of individuals who show reactions after repeated transfusions might compare fairly closely with the percentage of persons who would show symptoms of hay fever after repeated exposures to massive doses of ragweed pollen.

So where an individual must receive repeated blood transfusions and we make use of this knowledge concerning the Rh factor we may be able to avoid reactions or even deaths. Also, if the Rh factor of both parents is known we may be forewarned and forearmed against erythroblastosis fetalis. This knowledge is more valuable if supplemented by a careful history concerning any past allergic reactions in the persons involved.

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# X-Ray In Evacuation Hospitals*

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McALESTER, OKLAHOMA

I wish to present a brief outline of the organization and operation of the x-ray department in an evacuation hospital in the European Theatre, discuss some of the problems encountered, and cite a few case histories.

My unit was the 109th Evacuation Hospital, attached to the Third Army. We were in continuous operation during General Patton's five big campaigns on the Continent. Over 25,000 patients were admitted to our 400 bed hospital from D-Day to our inactivation in Czechoslovakia; 11,000 patients required x-ray studies. The evacuation hospital was the first hospital in the chain of evacuation from the fluid front lines where x-ray services were available to the soldier and the field medical officers.

The personnel of the x-ray department, as established by the Table of Organization, consisted of one medical officer, three technicians and a clerk. This set-up was inadequate in combat when the x-ray department was required to function continuously. Many of the x-ray departments acquired an additional medical officer and six to ten additional technicians or assistants, employing prisoners of war when available. I was unable to obtain a medical officer, but did acquire two additional enlisted men for our x-ray department. My duty hours were, therefore, determined by the volume of work in the hospital.

The most efficient x-ray team during combat consisted of four men. One technician set the portable x-ray in position and controlled the exposures. The second technician handled the cassettes and positioned the part to be x-rayed. The third technician processed, washed and dried the x-ray films and assisted the medical officers in their wet film readings. The clerk made daily entries, printed the identification slips for each individual film, filed the films of soldiers returned to duty and distributed the films of soldiers being evacuated to their respective wards or to the evacuation officer.

The equipment in the x-ray department consisted of:

(1) Two mobile Picker x-ray units with air cooled tubes. One of the units was used with the Westinghouse portable fluoroscopic table and screen.

(2) Westinghouse 5 gallon developing-fixing film tank with cooling-heating unit and an auxiliary wash tank, all housed in a portable dark room tent 9 x 12 feet.

(3) Two small gasoline generators to activate the x-ray machines and dark room equipment, placed outside the ward tent.

The civilian roentgenologists during the war were handicapped by the lack of sufficient x-ray films and an increased case load. The evacuation hospitals obtained all the x-ray films they required and desired, but we were unable to obtain x-ray accessories. We operated our x-ray department almost through the entire war with six cassettes, two of each standard size, and six corresponding film hangers for the three sizes of film. The exposed films were processed in the small, stuffy, hot, dark room. Wash water for films was delivered to us by our unit 700 gallon water tank every second to fifth day, depending on the source of the available water supply. The auxiliary wash tank in my unit was outside the dark room tent which permitted routine wet film readings. We did not have a regular film dryer, but we improvised drying units, built film racks which were placed near the stove, or we suspended films on wires above the stove and thus dried our films rapidly.

Fluoroscopy for localization of foreign bodies was very seldom employed in the evacuation hospitals of the Third Army. When we functioned as a station hospital, the surgeons occasionally reduced their fractures under fluoroscopic control. In the months before and after the Belgium Bulge, I employed the fluoroscope often on soldiers with peptic ulcer syndrome. The x-ray findings in the majority of these patients were negative, so that these soldiers were able to return promptly to their combat units. If the medical officer in charge of the x-ray department were not competent to do gastro-intestinal work, these soldiers were evacuated to the general hospitals for such diagnostic study and the soldiers were thus lost to their units for several weeks.

*Delivered before the Section on Dermatology and Radiology, Oklahoma State Medical Association Annual Meeting, May 3, 1946.

Superficial x-ray therapy in evacuation hospitals was discouraged by the army. The primary reason was that many of the officers were not trained in therapy. Second, machines were not calibrated in "r" units. I employed x-ray therapy for acute skin infections and skin lesions only on the personnel in our own organization or attached units. We had many cases of gas gangrene in our evacuation hospital that I would like to have treated with the mobile x-ray unit, but the army surgeons accepted the experimental findings of a group of American doctors who reported that x-ray therapy applied to B. Welchi organism in vitro was not of therapeutic value. They disregarded the mass of clinical evidence, the findings of Dr. Kelley and others, who showed that x-ray is the most valuable agent in the treatment of gas gangrene.

I would like to describe the operation of the x-ray department in an evacuation hospital during the periods of battle activity. The ambulances deposited their wounded at our receiving ward, often as many as two hundred casualties a day. The ward surgeons requisitioned x-ray studies on their first examination of most casualties in order to better determine the treatment and priority for surgery and to make assignments of these patients to the proper surgical teams. The assigned litter bearers therefore continued their trek back and forth from these wards to the x-ray department, until all x-ray requests were filled. During such periods of battle activity, sixty to eighty severely wounded patients, many with multiple injuries, would be x-rayed within a twelve-hour period.

There were several other factors other than the volume of work that made the x-ray technique and film interpretation of battle casualties more difficult than in the Army General or civilian hospitals. The wounded soldier was usually brought into the x-ray department before his blood stained, debris covered uniform was removed. X-ray studies were made with the patient on the sagging litter, through his clothes and the fracture immobilization device. Often the soldier had multiple injuries that prevented proper positioning of the part to be x-rayed. My technicians became very adept in angulating the stretcher and patient and tube in such cases to obtain a satisfactory roentgen study. They also became efficient in deciding the minimum number of x-ray exposures and views that were needed for the proper diagnosis and treatment of the patient in the evacuation hospital.

The following are examples of special technique that we necessarily employed in our evacuation hospital:

(1) Hemo pneumothorax, rupture of hollow viscus, or rupture of diaphragm were often diagnosed from x-ray films taken with patient in lateral decubitus position.

(2) Fractures of mandible, bones of the face and the skull were often made with the patient lying on his back, the tube directed from the side and below the stretcher.

(3) Lateral views of extremities for fractures and missiles, and lateral views of cervical, dorsal and lumbar spines were often necessarily taken with the tube in a horizontal position, rather than move the patient. Elevating the injured limb or arching the spine above the handles or metal parts of the stretchers was often difficult and time consuming.

(4) We resorted to the use of tangental views often to determine whether a missile was in the scalp or within the skull; in the lateral abdominal wall or within the peritoneal cavity; in the lateral chest wall or within the pleural cavity, etc. Stereoscopic equipment was not available in evacuation hospitals.

(5) Neither was Bucky Diaphragm available. Satisfactory diagnostic films were obtained with Lysholm grid.

(6) The unpredictable course of a missile demanded x-ray studies far from the site of entrance. For example, if missile entered shoulder, the dorsal spine or chest was x-rayed. If the missile entered the upper thigh, the abdomen was included in the x-ray studies.

The technique that I employed in our unit was that advocated by Mr. Fuch of Eastman Kodak Company; that is, the optimum voltage technique, the milliampere seconds being the variable. This technique is especially suited for rapid roentgen studies as required in the evacuation hospital. I continue to use this technique often in my practice in McAlester where I am in charge or four different x-ray departments.

The roentgenologist in an evacuation hospital was required to know x-ray technique. A roentgenologist can fool his colleagues in private practice about his x-ray technical ability, but he cannot deceive his army technicians who expect their medical officer to know all about x-ray and therefore consult him freely and often on technique. The medical officer who did not know technique rapidly lost the confidence and respect of his enlisted men, and the control of his department.

## CASE HISTORIES

1. X-ray studies of the base of the skull of a twenty year old soldier revealed a 2 x 1 cm. missile projecting into the medial edge of the left jugular foramen. The site of en-

trance of the missile was below the left inferior orbital rim. The soldier experienced pain on flexion of neck, deviation of tongue, syncope in upright position, slow unstable pulse, and a low unstable blood pressure. The neuro surgeon obtained a good exposure of the area by cutting sterno-mastoid muscle, but he could not locate the missile. The x-ray was brought into the operating room. The film showed the guide forceps on atlas instead of base of skull. The missile was found as diagnosed by x-ray. This was the first and only occasion that x-ray was carried into operating room in our hospital.

2. From routine A.P. and Lat. views of the chest of a battle casualty I localized an intact 45 cal. bullet within the right sterno-clavicular joint. I was called to the operating room to justify my diagnosis after the surgeon failed to find the bullet. The bullet had entered high on the left lateral side of neck and its path was traced behind the manubrium. I requested that the surgen re-expose the right sterno-clavicular joint and that he open the capsule of this joint. The bullet was found imbedded within the joint in articulating surface of the clavicle.

3. A 21 year old S/Sgt. entered the hospital with a one cm. wound to the left side of his neck at the level of thyroid cartilage, and another 3 cm. wound to left of spine of second dorsal vertebra. The ward surgeon considered these two wounds due to a penetrating missile. X-ray of chest did not reveal any missile or pneumo hemothorax. Two days later the soldier developed athetoid movements and disorientation and on the fourth day paralysis of lower extremities. He died on the fifth day. The spinal cord injury was not suspected early because the ward surgeon was mislead by the course of the missiles. Post mortem examination revealed a fracture of arches and laminae of left side of second and third dorsal vertebrae with depression of the cord. A niche in the dura and hemorrhage was found at the fracture site, but the cord was intact.

4. A soldier assigned to a tank unit entered the hospital with paralysis below level of second lumbar vertebra. The site of entrance of missile was on right side of upper back. The missile was located by x-ray outside the spinal canal over the first sacral vertebra. X-ray of spine revealed a complete absence of entire right lamina and inferior articular process of third lumbar vertebra. The cord was found crushed at level of twelfth dorsal. Review of films showed that the absence of lamina of the third lumbar was due to developmental anomaly.

5. An irregular 1.5 cm. missile fractured right eighth rib posteriorly, penetrated chest, and was located by x-ray in the left posterior axilla at level of sixth rib. The soldier was observed for two days in clearing hospital because of bilateral paresis of lower extremities which disappeared on admission. X-rays revealed bilateral pneumothorax, both lungs collapsed equally about 50 per cent, small amount of fluid at bases. Fluoroscopy revealed elevation of left diaphragm in inspiration. In expiration the diaphragm and fluid in both sides of chest were at same level. Aspiration of over 2,000 cc of air from right chest produced no change whatever in x-ray picture or patient's condition. The soldier had a perforation in posterior mediastenum. He recovered.

We had a similar case the next day. In this case the missile severed the cord at the level of the twelfth dorsal vertebra.

6. A 20 year old well developed soldier entered the hospital because of an erysipelas-like rash in right shoulder and axilla 24 hour duration. History of mild injury three days prior to admission. A single circular erythematous patch about 3 cm. in diameter was noted on his left arm. His temperature was 104°. He was placed on sulfa therapy. Within twelve hours he developed generalized petechial rash, athetoid movements and became semi-comatose. Some stiffness of neck was now observed. Temperature 105°, blood pressure 60/40. Patient died within 24 hours. Post morem revealed petechial hemorrhages in all organs of body and body cavities. The generalized petechial hemorrhage in adrenals explained the rapid fall in blood pressure and shock.

The designated name for this type of meningococcemia is Waterhouse-Friedrich syndrome.

## CONCLUSIONS

I have recorded in my personal files, a few hundred case histories of unusual injuries and clinical findings resulting from missiles. It is difficult to convey to you the medical-surgical interest in these cases, or the emotional reaction that I experienced by contact with these battle casualties who came to our hospital within a few hours after they received their injuries. In this dramatic setting, x-ray work in a field hospital was always interesting, and the results of our efforts were most gratifying.

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Joy is more divine than sorrow; for joy is bread and sorrow is medicine.—*Henry Ward Beecher.*

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Silence is the perfectest herald of joy: I were but little happy if I could say how much.—*Shakespeare.*

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Nothing is so envied as genius, nothing so hopeless of attainment by labor alone. Though labor always accompanies the greatest genius, without the intellectual gift, labor alone will do little.—*B. R. Haydon.*

# Clinical Pathological Conference

*University of Oklahoma School of Medicine*

*Presented by the Departments of Pathology and Medicine*

ROBERT H. BAYLEY, M.D.—BELA HALPERT, M.D.

OKLAHOMA CITY, OKLAHOMA

DR. HALPERT: The patient whose story we are presenting today gave opportunity for very thorough clinical study. Because of this, clinicopathologic correlation should be of more than usual interest. Dr. Bayley will present the clinical aspects of this case.

PROTOCOL

*Patient*: O. B. D., white female, age 40; admitted August 6, 1945, died November 2, 1945.

*Chief Complaints*: Weakness, loss of weight, and fever.

*Present Illness*: The patient was well until September, 1944, when, following the removal of several teeth, she developed weakness and pain in the joints of her arms and legs. The joint pain was severe; however, she noticed no redness, swelling, or increased heat. One month later she noted that she was having a continuous low-grade fever. She went to the Duncan Hospital at this time and has been bedfast since. During hospitalization she heard her doctors remark that her heart "was bad". The weakness, low-grade fever, and lassitude have continued to the present time. She lost about thirty pounds of weight. There has been dyspnea and precordial aching with some palpitation. There is no history of cough, expectoration, hemoptysis or pleurisy. The remainder of the history is noncontributory.

*Past and Family History*: Noncontributory except for the usual childhood diseases and one miscarriage.

*Physical Examination*: The patient was emaciated and had many carious teeth. Cooperation was poor. The skin was very loose and dry and presented a very light brown pigmentation. The pupils were equal in size, reacted to light and accommodation. There were a few small petechial hemorrhages in the conjunctiva. The fundi were negative. There was slight cheilosis. Mucous membranes exhibited moderate pallor. There was no lymphadenopathy. The lung fields were clear. The cardiac apex impulse was approximately 8 cm. to the left of the midline and of increased forcefulness. A rather loud apical systolic murmur was heard. Blood pressure was 115/65 and pulse rate 100. The liver was not palpable. The spleen extended two finger breadths below the costal margin on deep inspiration; its edge was firm, its surface smooth and non-tender. There was no edema of the feet or ankles. Fingers exhibited moderate clubbing. Examination of genitalia revealed a cervical and vaginal discharge, also a purulent urethral discharge. A cystocele and rectocele were present.

*Laboratory Data*: During her two months hospitalization, urinary specific gravity varied from 1.008 to 1,014, proteinuria from 0 (once 3 plus). No red blood cells were found. The red blood count ranged from 2.2 to 3.7 million; hb. varied from 5 to 9 grams. Leukocytosis was usually present, averaging 16,000 with 82 per cent polys. Spinal fluid was negative. Three Mazzini tests were: 3 plus, 2 plus, and negative. Two Kolmer reactions were negative. Cervical and urethral smears revealed intracellular Gram negative diplococci on four occasions. On two additional occasions mixed flora were found. On one occasion a culture of gonococci was negative. Two blood cultures were negative. An electrocardiogram was reported as negative. A requested sound tracing was not done because the apparatus was not available. It was stated, however, that there was a 3 plus accentuation of the mitral first sound, followed by 3 plus systolic murmur, and preceded by a diastolic rumble. An aortic diastolic murmur was also present along the left border of the sternum at the third and fourth I.C.S. There was no evidence of free aortic insufficiency, rheumatic. On the eighth hospital day, penicillin was given, 50,000 units every three hours for three doses, then 25,000 units every three hours for five doses. Sulfathiazole, which had been started two days before the penicillin, was discontinued on the 20th

day. A psychiatric consultant stated that no psychopathic condition was present, but that the negativistic reaction was probably a part of her gradually developed personality since childhood. On the 3rd hospital day penicillin was given again, 25,000 units every three hours for 72 hours. On the 57th hospital day an arterial blood culture showed growth of a nonhemolytic staph aureus in both tubes. X-ray taken on August 8, 1945, showed heart and lungs normal except for quite a prominent pulmonary conus. A second x-ray, taken on September 26, 1945, suggested cardiac enlargement and pulmonary congestion.

*Clinical Course*: Low grade fever continued with septic spikes, occasionally up to 103 degrees. She was often uncooperative and refused medication on several occasions. Sulfathiazole was started August 12, 1945. On August 14, 1945, penicillin was given which was repeated at intervals through the course of the disease. On November 2, 1945, the patient complained of severe abdominal pain. Examination showed marked abdominal distention, rigidity and absence of borborygmus. A Wangensteen tube was ordered. There was blood in the stools and she vomited blood once. She died on November 2, 1945.

CLINICAL DIAGNOSIS

DR. BAYLEY: Our case today is that of a white female, aged 40, who was admitted to and died on the Medical Service. Her chief complaints of weakness, loss of weight and fever immediately suggested some infectious process. Tuberculosis would be a distinct possibility in view of the general appearance which the patient presented and the rather insidious onset of symptoms. The onset of her present illness was characterized by joint pains and this was soon accompanied by low grade fever. Such an onset suggests undulant fever, rheumatic fever, or perhaps rheumatoid arthritis. She is a little advanced in age for this latter, but we do sometimes see rheumatoid arthritis beginning at this age. During her initial hospitalization she overheard her doctor remark that she had a bad heart. It is difficult to interpret such a statement from the patient. Knowledge of the exact status of things at this point would be very helpful. This does further our consideration of rheumatic fever, however. We know that patients with subacute bacterial endocarditis have insidious onset, loss of weight, fever and joint pains, so that we must include this in our differential diagnosis. The fact that her joints were not swollen or reddened would be evidence for bacterial endocarditis, rather than active rheumatic fever. There is also a history of some precordial pain. This is difficult to evaluate but would be compatible with either condition. There is no history of hemoptysis. The patient's past history does not contribute much of value.

Before we review the physical examination it might be interesting to anticipate some of the possible findings. Since we have considered tuberculosis, we should expect to examine the lungs thoroughly — similarly with the heart since we are considering rheumatic fever also. As a rule, cardiac murmurs which appear during the course of active rheumatic fever disappear by the end of the acute attack. It may be six months later before "residual murmurs" develop. We should carefully examine the joints for redness, etc., because this would definitely point toward rheumatoid arthritis. If this were rheumatoid arthritis, we should find little change in the heart. Splenomegaly does not accompany rheumatic fever, but occasionally does characterize rheumatoid arthritis (Felty's syndrome). The patient had moderate anemia. This is often seen in rheumatic fever and is an invariable accompaniment of subacute bacterial endocarditis. The loose dry skin may be interpreted as a manifestation of dehydration. The diffuse, light brownish pigmentation would fit in with subacute bacterial endocarditis. Conjunctival hemorrhages (petechial), such as were recorded here, should be carefully inspected because often examination may reveal a white center. Such lesions may be found in the occular fundi when they appear nowhere else. These, of course, would represent tiny septic infarcts and would be pathognomonic of subacute bacterial endocarditis. Now to consider the heart —this was not appreciably enlarged although pulsations were of increased forcefulness. As a rule, in subacute bacterial endocarditis, the heart shows little enlargement. It is an interesting point that those patients who have repeated attacks of rheumatic fever (and cardiac enlargement) are the very group who do not develop bacterial endocarditis. Dr. Levine has pointed out that the onset of subacute bacterial endocarditis after the last attack of rheumatic fever is, on the average, about 17 years. Subacute bacterial endocarditis has been noted to occur, however, during an attack of rheumatic fever. Here we are up against the problem of differentiating between rheumatic fever and subacute bacterial endocarditis. We know that in both cases there are joint pains and fever. Those patients having rheumatic fever usually have striking cardiac enlargement; with subacute bacterial endocarditis there is no such enlargement. In rheumatic fever, splenomegaly is not characteristic. The splenomegaly which this patient exhibited is typical of subacute bacterial endocarditis. Here in examination of the heart there was a diastolic and presystolic murmur. The apical systolic murmur may have been hemic in origin. The blood pressure was 115/65 and the pulse rate 100.

All of these findings point toward bacterial endocarditis. We have also the history that just before onset of this present illness the patient had several teeth removed. Not infrequently the extraction of infected teeth causes a bacteremia which, in turn, leads to subacute bacterial endocarditis. I saw this patient clinically; the diagnosis of subacute bacterial endocarditis was entertained throughout her hospital course.

Regarding the laboratory findings, the leukocytosis observed is quite compatible with bacterial endocarditis.

I believe that the positive Mazinni tests were not significant because of the septic state. Subacute bacterial endocarditis is rarely superimposed upon a luetic lesion, but this can occur. The vaginal and urethral discharge, with demonstrable gram negative intracellular diplococci should make the diagnosis of gonorrhea — this infection I believe was incidental. Gonococcal endocarditis does rarely occur, but usually involves the pulmonic valves. All evidence here points to involvement of the left heart. Venous blood cultures were negative upon two occasions. In most instances subacute bacterial endocarditis is limited to the left side of the heart. Because of this, bacterial emboli are filtered out in large measure before ever reaching the venous system. Often in these patients venous blood cultures will be negative whereas arterial blood cultures, taken at the same time, may be positive. In this case a culture of arterial blood was positive for nonhemolytic Staph. aureus. This is a very unusual organism to cause subacute bacterial endocarditis. The same organism was recovered in two culture tubes which would be strong evidence that this was not a laboratory contamination. It is possible, however, that the blood was contaminated at the time it was drawn. At any rate this lends much additional support to our diagnosis. To consider the precise character of this lesion in the heart we must consider again the physical findings. From the type of cardiac murmurs heard it was my opinion that this patient had vegetative lesions of both the mitral and aortic valves. On the basis of this diagnosis, one expects a wide variety of embolic phenomena. It may have been on the basis of tiny cerebral embolic lesions that psychic disturbances were manifest.

Regarding treatment it must be said that in the light of recent clinical studies the amount of penicillin which this patient received was certainly very small in comparison with the relatively enormous doses necessary to effectively treat this condition.

## CLINICAL DISCUSSION

QUESTION: Do you think that, from the clinical evidence, the rheumatic process was active at the time of death?

DR. BAYLEY: That is possible, but it is unusual in subacute bacterial endocarditis and there is no clinical basis for a diagnosis of active rheumatic fever.

QUESTION: Could some anomaly, such as a bicuspid aortic valve, rather than a rheumatic lesion, have been the underlying basis for this bacterial endocarditis?

DR. BAYLEY: I doubt it. In the first place, rheumatic fever is much the more common predisposing factor and secondly, we have evidence that suggests involvement of the mitral valve in addition to the aortic valve.

DR. HALPERT: Perhaps we should review the x-ray studies on this patient.

DR. SHRYOCK: These roentgenograms show no cardiac enlargement although there is a marked fullness and prominence of the pulmonary conus. There is also increased hilar vascular markings in the lungs.

DR. BAYLEY: Would these findings be compatible with bronchopneumonia?

DR. SHRYOCK: I can see no definite evidence of cellular infiltration.

QUESTION: Is the pulmonary osteo-arthropathy related to rheumatic fever?

DR. BAYLEY: No, rheumatic fever does not cause clubbing of the fingers. Subatcute bacterial endocarditis, on the other hand, frequently leads to this.

## ANATOMIC DIAGNOSIS

DR. HALPERT: At necropsy this woman showed evidence of marked emaciation; she was 61 inches tall and weighed less than 100 pounds. The skin presented purple blotches which faded on pressure. No petechiae were noted. The abdomen was moderately distended and there was approximately 400 cc. of bloody fluid in the peritoneal cavity. A layer of fibrinopurulent exudate covered the surface of the liver. The mesenteric vessels were firm and an embolus occluded the superior mesenteric artery for a length of 4 cm. There was an aneurysm of the inferior mesenteric artery a few cm. from its origin. The sudden occlusion of the superior mesenteric artery had resulted in infarction of a large segment of ileum. This portion of bowel was distended with gas, discolored reddish brown and covered with fibrinopurulent exudate. No definite point of perforation was observed. There is little question but that the peritonitis resulted from passage of bacteria through the wall of this markedly altered ileum. Both pleural cavities contained serous fluid, the left approximately 100 cc., the right, 800 cc. The heart was slightly enlarged, the result of dilation. It measured 12 cm. from base to apex and 12 cm. across the base; it weighed

325 Gm. The mitral and aortic valve cusps exhibited nodular areas of fibrous tissue at the line of closure and there was moderate thickening of the valves themselves. Friable granular vegetations were present on both the mitral and aortic valves. The myocardium was light tan and flabby, particularly on the right side. The lungs were moderately increased in weight and there was an area of lumpy consolidation in the right upper lobe with some hemorrhage. The spleen, although palpable during life and estimated to be quite large, weighed only 220 Gm. The liver was somewhat decreased in size and weighed 1100 Gm. The kidneys weighed 100 Gm. each. No areas of interstitial hemorrhage were discernible. Minute foci of hemorrhage were apparent on microscopic examination however, and the picture was actually that of embolic glomerulonephritis. Cultures from the valvular vegetations were not taken at necropsy. In the spleen there were numerous scars which were quite characteristic of old infarcts. There were no recent infarcts.

DISCUSSION

QUESTION: What was the extent of the gonorrheal infection?

DR. HALPERT: This was limited to the cervix and was minimal at time of death. It is probable that the penicillin therapy had resulted in an almost complete cure.

QUESTION: What caused the splenomegaly which was so evident clinically?

DR. HALPERT: This is readily accounted for on the basis of septicemia. Occasionally in cases of this sort the spleen becomes smaller in the terminal stages.

DR. BAYLEY: In people with subacute bacterial endocarditis the temperature goes down and the spleen reduces during treatment, but when therapy is discontinued or becomes ineffective, the spleen enlarges again.

DR. HOPPS: At the University of Chicago we were quite impressed by the rather high percentage of persons with subacute bacterial endocarditis whose onset of illness followed tooth extraction. Just recently, experimental studies carried out by the Zollar Dental Clinic demonstrated that transient bacteremia occurs in almost 100 per cent of patients following the extraction of a tooth. Particularly, in patients with a history of previous rheumatic fever, I believe that sufficient sulfonamides should be given to maintain a rather high blood level during the actual time of tooth extraction and for a period of twelve to twenty-four hours afterwards.

DR. BAYLEY: I agree with Dr. Hopps except that I believe penicillin would be the drug of choice. This would necessitate, however, an intramuscular injection.


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# Special Article

## DEAN GRAY

On September 1, Dr. Jacques P. Gray entered upon his duties as Dean of the Medical School. He succeeded Dr. Wann Langston who has served in the capacity of Acting Dean from the time of Dr. Tom Lowry's death.

Dr. Gray is eminently fitted for this position.* Through both education and experience he has become a seasoned administrator. He comes to us with a splendid reputation, academic and administrative, in medical education and public health.

In the last analysis the medical school is the property of the people, the taxpayers. The law says there are two kinds of property, real and personal. To the doctor this ownership should be genuinely personal. While this applies particularly to the Alumni, every doctor in the state should manifest a real interest in the medical school and should exact a certain amount of interest and consideration from the medical school. Working together, the Dean, the faculty and the doctors of the state should see that medicine in Oklahoma comes into its own and that it gives to the medical profession and the people all that the taxpayers have a right to expect, plus a gratuitous bounty in keeping with traditions of medicine.

With the above in view it may be said that every doctor in the state should stand four square behind the Dean. With the present enthusiasm of the Alumni and the growing interest of the public in medical research, Dean Gray comes at a propitious time. The medical school is part of a great university; it does not belong to Oklahoma City. Every doctor in the State should become vitally interested in medical education and scientific research and make his individual contribution by helping the Dean hold the torch high.

*Dean Appointed, Journal of the Oklahoma State Medical Association, page 376, September, 1946.

# THE PRESIDENT'S PAGE

## PROFESSIONAL COURTESY

Recently my attention was called to an experience a physician had with a large Clinic. He wrote for an appointment for himself and wife, so that they might have a check up, designating a date twelve days from the date of his letter. In reply he was told he could have an appointment three months from the date the reply was written, unless it was an emergency.

This particular physician was well known to them as he had sent many patients to the Clinic and had himself been a patient. They showed lack of professional courtesy in his case when they did not take into account the fact that he also was a physician.

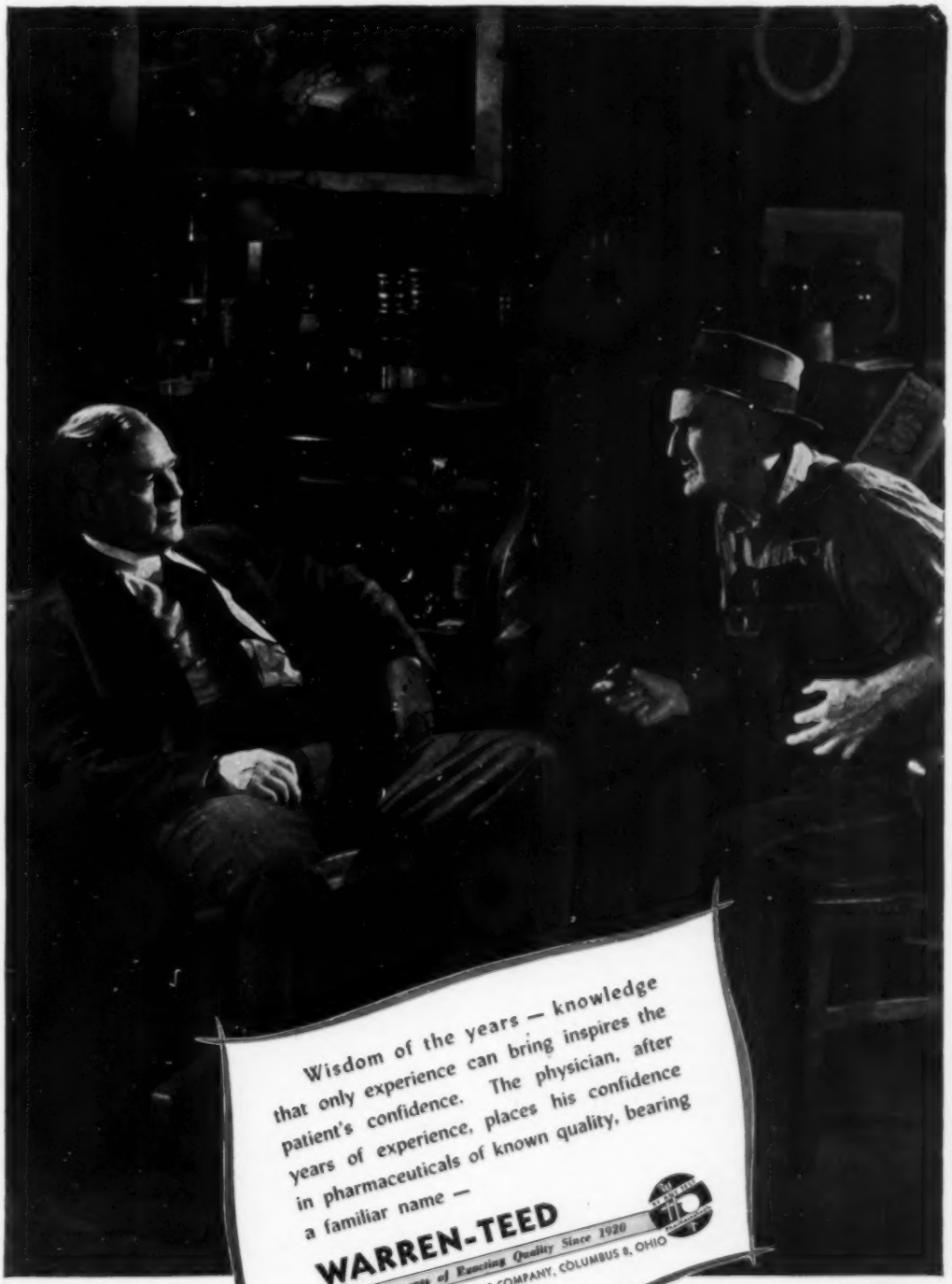
Then there is the case of the physician who will accept a patient, who is under treatment by another physician, when the patient has not been dismissed by the physician or paid for his services.

It sometimes happens that after a physician is called in Consultation he will accept the patient for treatment without the original physician's knowledge or consent.

All of these constitute lack of professional courtesy and should not be practiced at any time.

L. C. Kuykendall

*President.*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# The JOURNAL Of The
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# EDITORIALS

### *THE FALL CLINICAL CONFERENCE*

The Oklahoma City Clinical Society has consummated plans for its great Clinical Conference on October 28, 29, 30, 31.

The first announcement was in the September issue of the Journal, but this late appeal is intended for those who have not "red-lettered" the dates. No honest, ambitious practitioner can afford to miss this opportunity for post-graduate study. Never in the History of Medicine has the need of such refresher studies been so urgent.

Henry Christian has said, "There were two great groups of physicians; those who are learning and those who are forgetting." Will you be there or will you be forgetting?

---

### *RESOLUTION ADOPTED BY A.M.A.*

At the American Medical Association meeting in San Francisco, July, 1946, the House of Delegates passed the following resolution:

**RESOLUTION ON STATEWIDE MENTAL HYGIENE AND MENTAL DISEASE PROGRAM***

"Dr. G. A. Woodhouse, Ohio, presented the following resolution, which was referred to the Reference Committee on Medical Education.

"WHEREAS, there is an urgent need in most of the states for well organized and adequately financed mental hygiene programs, for research activities in the field of mental disease and for improved institutional care of the mentally ill; and

"WHEREAS, the medical profession should give increased leadership and support to such activities; therefore be it

"RESOLVED, that each state medical association be requested to take the lead in the development of an adequate statewide mental hygiene and mental disease program and to cooperate with other groups in stimulating public support in order that sufficient funds may be secured for the proper operation and maintenance of such activities."

This resolution should have careful consideration by every doctor in Oklahoma. No doctor can escape the responsibility of dealing with the mentally inadequate and the psychologically disturbed. Every patient who seeks medical advice presents a mental threshold which the doctor must measure and beyond which he must go with analytical perception and precision.

. . . *Proceedings of House of Delegates, American Medical Association, San Francisco, July, 1946.

Considering the rapid increase in mental diseases and the recent agitation concerning the short comings of institutional management throughout the United States doctors should initiate and actively support a well balanced program for research in mental hygiene and improved institutional care of the mentally ill.

## *RESEARCH*

Soon our boys will go out to raise funds for the University of Oklahoma Research Foundation. When they do we must shell out. The spirit of scientific investigation is more than 2000 years old. We do not need more time to think it over. We must set the example and through our interest and generosity we should not only reach down in our jeans but jar loose some of the wealth of Oklahoma.

After 20 centuries Oklahoma has a chance to come into her own. This is no time for doctors to hesitate. The eyes of the world are upon us. If giving means a sacrifice, it should be remembered that the money will be used to purchase sacrificial service for the purpose of controlling disease and saving life. Who ever knew a bonafide scientific investigator in the field of medicine who was not underpaid.

This is our chance to tie up the general practitioner with the worker in pure science through the medical school teachers as middle men. Only in this way can the medical profession and the medical school keep faith with the people.

Would that this editorial had the fire and thunder from Sinai or at least the persuasive power of the Sermon on the Mount. Even though the stimulus is wanting, let's give.

## *PUBLIC HEALTH IN OKLAHOMA*

Public health in Oklahoma is now 56 years old and has made great strides during the past year. From its humble beginning, as provided by the first territorial legislature in December of 1890, the State Department of Health has grown to assume a protective influence for all citizens of the state.

During the early territorial days, the Superintendent of Public Instruction served as ex-officio president of the Board of Health and was assisted by two staff members, a vice-president who served without pay and a Superintendent of Public Health who received $500 annually for recording the work and acts of the health department.

At this time, the Board of Health was authorized to make and enforce needful rules and regulations for the prevention and cure of disease. In this connection, licenses were issued permitting doctors to practice medicine in the territory.

In early statehood, the Oklahoma State Department of Health was impowered to establish quarantines, to remove the carcasses of dead animals, to condemn and remove any impure or diseased articles of food offered for sale, and to prevent the spread of any contagious, infectious or malarial disease among persons or domestic animals.

The influence of public health has grown. To understand how much it has grown we need only compare the appropriation of $33,600 which was granted public health officials in 1910 to the last appropriation of $549,752 which was granted for the fiscal year ending June 30, 1947. Even this increased appropriation is not sufficient to undertake all authority delegated to the State Department of Health by the twentieth session of the Oklahoma State Legislature.

During the past few years many acts pertaining to public health have been adopted as a culmination and recognition of over 50 years of work by officials of the State Department of Health.

Recent legislation required an examination for syphilis prior to the issuance of a marriage license. Another act created the Board of Health, which is impowered to appoint a Commissioner of Health.

Frozen food lockers are now subject to inspection and regulation, as are all bedding manufacturers in the state. Serological blood tests for syphilis of pregnant women is now a state requirement; and on July 1, the State Department of Health undertook to inspect and license all hospitals and related institutions now operating in the state.

The legislature appropriated $231,000 for the fiscal year ending June 30, 1947, to be used in financing county health departments; and the State Department of Health was authorized to accept grants of money, personnel and property from federal agencies to promote and carry on in Oklahoma a program of public health, maternity and infancy care, venereal disease control, tuberculosis control, malaria control, industrial hygiene and sanitation, urban and rural sanitation and other phases of a general public health program.

Senate Bill 153 provided authority for school districts, boards of education, cities and towns to furnish funds to assist in the operation of local health departments. For the first time in the history of the state money was appropriated for the State Department of Health Tuberculosis Control and Industrial Hygiene Divisions. Tuberculosis Control received $10,000 annually and $5,000 was appropriated for Industrial Hygiene. In addition, Venereal Disease Control was allotted $20,000 for each fiscal year, or an increase of $10,000 annually.

The people of Oklahoma should take notice of the fact that the legislators have recognized the importance of promoting better health and constructed legislation to that end. Much credit for the enactment of these laws belongs to Governor Kerr, who outlined the public health needs in his message to the legislature.

Our thanks should also go to the capable public health workers who have overcome seemingly insurmountable obstacles to render good and efficient service, and to the House of Delegates of the American Medical Association and the Oklahoma State Medical Association for their resolutions endorsing and advocating the establishment of full time local health departments to serve every area and unit of population.—Grady F. Mathews, M.D.

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## *THIS TOTTERING WORLD*

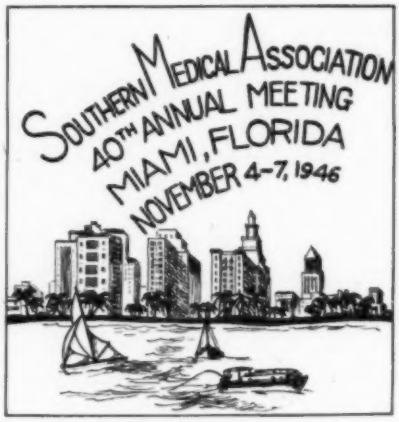
Atlas was having a difficult time holding the world aloft until Socrates taught the world to look to reason for stabilization and medicine under the stimulus of Hippocrates, and Socrates initiated the age of enlightenment and helped to bring individual freedom in thought and action.

Under Greek influence, medicine for the first time was free and democratic. With the exception of the mad, and the morons, people learned to keep their heads, their own little worlds, squarely on their shoulders. This individual balance helped to secure collective equilibrium, thus enabling Atlas to hold the world in its appointed place.

The coming of Socialism and Communism in this modern era has robbed Atlas of the steadying influence which came through individual balance and we feel the world swaying on the Titons tired shoulders. With North America looking upon socialistic trends as the way to redemption and the bureaucrats standing ready to rob Atlas of his main stabilizing force — medicine as a free enterprise — we may expect the crash unless we "Wake up America".

If Atlas, created for the task, is having a hard time with the wobbly cockeyed world, how can Mr. Altmier, the Surgeon General of the Public Health Service and their bureaucratic cohorts hope to cope with it after knocking the last prop out.

In the eyes of the thinking citizens of the United States, they are either too busy to look the proposed adventure squarely in the face or they are the world's prize egoists. Perhaps some of the cortex they are counting on may prove to be cheap cork. Shall we be damned by their exaltation or shall we act upon the knowledge at hand and put them in their places.


SCIENCE AT THE NATION'S PLAYGROUND — a top scientific medical meeting amid delightful tropical surroundings with every known recreational facility available — Southern Medical Association, Miami, November 4-7. The Southern Medical Association meetings always have been and always will be the ESSENTIAL medical meetings IN and FOR the South. In its twenty-one scientific sections, the four general clinical sessions, the general public session, the four conjoint meetings and the scientific and technical exhibits, in a streamlined program, one will get the last word in modern, practical, scientific medicine and surgery. And after Miami, Havana and/or Nassau.

**REGARDLESS of what any physician may be interested in, regardless of how general or how limited his interest, there will be at Miami a scientific program and recreational facilities to challenge his every interest and make it worth-while for him to attend.**

ALL MEMBERS of State and County medical societies in the South are cordially invited to attend. And all members of state and county medical societies in the South **should be** and **can be** members of the Southern Medical Association. The annual dues of $4.00 include the Southern Medical Journal, a journal valuable to physicians of the South, one that each should have on his reading table.

**SOUTHERN MEDICAL ASSOCIATION**
Empire Building
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# RESEARCH FOR HUMANITY

**John H. Lamb, M.D.**

The Oklahoma Medical Research Foundation was formally incorporated on August 3, 1946, at a dinner held at the Skirvin Hotel. Mr. George Lundy of the firm of Marts and Lundy was a guest at this meeting, at which time he outlined plans for a campaign to begin early in October composed primarily of publicity the first two or three months, followed by an intensive financial drive in January or February.

The initiating group was selected by the trustees of the Alumni Association and is made up of twenty directors and twenty laymen. The physician directors were selected from three groups, the State Medical Association, the Alumni Association, and three representatives from the Faculty of the School of Medicine.

It has been an earnest desire on the part of those who established the Foundation to spread their influence over the entire State. Those who were placed on the initiating Board were those who have shown interest in the past several years. By-Laws of the Foundation state that there will be a yearly election, at which time those who have becomes members of the Foundation in the various groups by their contributions will have a vote to elect their respective directors on the Board. It is planned to make the Foundation a state-wide democratic organization so that the entire State will have an interest in Medical Research and the School of Medicine.

This organization has great potentialities, not only to interest the public in endowments for the School of Medicine, but by bringing expert Research Personnel to Oklahoma Medicine, thus medical educaton will be advanced one hundred fold.

It is felt that the motto "Better medical education today means better health tomorrow" would be an excellent theme for the campaign. At the incorporation meeting August 3 the following were elected as Directors of the Foundation:

LAYMEN: Henry S. Griffing, L. C. Griffith, C. R. Anthony, Edgar C. Van Cleef, W. R. Wallace, D. A. Harmon, Roy C. Lytle, Moss Patterson, C. A. Vose, Ancel Earp, Fred Jones, and Robert S. Kerr, of Oklahoma City; Fred L. Dunn, Ralph Talbot, Charles L. Follansbee, of Tulsa; H. B. Alspaugh, Duncan; J. Frank Buck, Shawnee; Doan Farr, Clinton; Clyde Muchmore, Ponca City; and W. Lee Woodward of Alva.

ALUMNI: W. Floyd Keller, M.D., and John H. Lamb, M.D., Oklahoma City; Ralph McGill, M.D., and Fred E. Woodson, M.D., Tulsa; William Finch, M.D., Hobart; John Carson, M.D., Shawnee.

OKLAHOMA STATE MEDICAL ASSOCIATION: Henry C. Weber, M.D., Bartlesville; O. C. Newman, M.D., Shattuck; Paul Champlin, M.D., Enid; Lewis J. Moorman, M.D., and Henry Turner, M.D., Oklahoma City; James Stevenson, M.D., Tulsa.

FACULTY: Everett S. Lain, M.D., Howard C. Hopps, M.D., and L. Chester McHenry, M.D., of Oklahoma City.

It is hoped that the entire medical profession will catch the vision and the possibility of this new organization and support it, not only financially, but by their influence on potential givers throughout the State. There have already been many interesting promises made by several potential donors and it rests with the medical profession to initiate this campaign by every doctor giving $100 or more to raise the $25,000 for the expenses of the first five months of the campaign.

Let's help Oklahoma Medicine advance.

---

# HOSPITAL CONSTRUCTION ACT

The State Commissioner of Health, Dr. Grady Mathews, gave a summarization of Public Law 725, commonly referred to as the Hill-Burton Bill at a meeting of the Woodward County Medical Society in Shattuck, Oklahoma, September 11.

The purpose of the act is to assist the States in planning for and providing the modern hospital and public health facilities needed in order that all people may be served. In order to be eligible to participate in the program, each State must prepare a State plan which conforms with the regulations to be prescribed by the Surgeon General, and approved by the Federal Hospital Council.

Construction application forms will be made available for distribution by the State agency when funds are appropriated by Congress. All applications for individual projects must first be approved by the State agency, and then forwarded to the Surgeon General for his approval.

Congress has authorized the appropriation of $75,000,000 a year for the next five years. *No money for construction has yet been appropriated.* Such an appropriation is not possible until Congress reconvenes. On the basis of the $75,000,000 authorization, Oklahoma will be entitled to an allotment of approximately $1,640,550 annually for the next five years, beginning with the fiscal year June 30, 1946. From this allotment a payment of one-third of the construction cost will be made to each individual approved project.

### Information for Each Application

1. Description of site.
2. Plans and specifications which must meet standards and regulations promulgated by both the State and Surgeon General.
3. Title to site must be vested solely in applicant.
4. Reasonable assurance that adequate financial support will be available for the construction of the project and for its maintenance and operation when completed. (This would seem to indicate that the amount of local money must be sufficient not only to meet at least 66 2/3 per centum of the construction cost, but there must be sufficient reserve to provide for operation and maintenance for at least two years.)
5. Applicant must agree rates of pay for laborers and mechanics will be not less than prevailing local wage rates.
6. Facilities must be available to all without discrimination on account of race, creed or color. (This indicates that in counties with an appreciable negro population, provision must be made for the care of this class of people.
7. Each hospital receiving funds under this law must make provision for those unable to pay therefor. (Perhaps the Surgeon General may in his regulations indicate the amount or per centum to be used for "those unable to pay". If not, you will no doubt need to determine this).
8. All institutions receiving funds under this law must be operated on a non-profit basis.
9. Applicant must agree to reimburse the Federal Government if, at any time within 20 years, the hospital is transferred to a person, agency or corporation not eligible to file application under this Act. Value at time of transfer to be determined by agreement or by action in U. S. District Court.

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## SOUTHERN MEDICAL VIA SPECIAL TRAIN

A Special Train to Southern Medical Association, Miami, Florida, and a side trip to Cuba will get under way in November, the details of which are being handled by Mr. Harry Kornbaum. The many doctors and their families who attended the A. M. A. in June will remember Mr. Kornbaum as having conducted the trip through the Northwest, making it a most pleasant and delightful tour.

### Transportation

For those who prefer to fly, it is anticipated that there will be a Special Plane leaving Oklahoma City at 5:00 A.M., November 3, making the return trip the afternoon of November 8.

Those going to Miami by train will leave via the Frisco, from Oklahoma City, November 1, 5:50 P.M., picking up Tulsa members of the party at 8:45 P.M. and arriving in Miami November 3 at 5:10 P.M. Hotel reservations for a maximum of 120 doctors and their wives have been made at the famous Robert Clay Hotel, located four blocks from the Convention Hall.

### Post Convention Tour to Cuba

A Pan-American Clipper will take members of the party who desire to go to Havana, Cuba, on a post-convention tour. The plane leaves Miami November 8, arriving in Havana after 90 minutes of flying time. Reservations are made at Cuba's "ultra" hotel, the Nacional de Cuba, for four days and three nights. The Clipper returns to Miami November 11, allowing ample time to board the Special Train and head up the seaboard coast for Jacksonville and New Orleans.

### Coming Home

The Special will pass through Jacksonville the morning of November 12, turning westward along the Gulf Coast and arriving in New Orleans the following morning, November 13. The party will enjoy a pleasant two-day stopover in New Orleans with sight seeing trips through the many places of interest in this picturesque old town.

At 8:50 A.M., November 15, the party boards the Special Train for the remainder of the trip home. Passing through Memphis at 7:20 P.M. of the same evening, we return to Tulsa November 16 at 9:55 A.M. and into Oklahoma City at 12:40 P.M.

### Reservations

The trip to Miami and side tour to Cuba, if elected, is on an all expense basis throughout, except meals. Individual reservations for the return trip for those who do not wish to make the post-convention trip or vacation in Miami, awaiting the return of the Special Train, will be made. There will be available upper berths, lower berths, and compartments. In making reservations, please specify in detail how you wish to travel and whether or not you desire to make the post-convention trip to Cuba. Time is somewhat limited; please make your reservations as soon as possible with Mr. Harry Kornbaum, Rainbow Travel Service, First National Building, Oklahoma City.

## PROGRAM FOR VETERANS MEDICAL CARE PROGRESSING RAPIDLY

As the Journal goes to press the Oklahoma State Medical Association's plan of cooperating with the Veterans Administration to render medical care to veterans is rapidly nearing completion.

Dr. John F. Burton, Chairman of the Committee, studying the plan, and Mr. Dick Graham visited the office of the Kansas plan in Topeka, Kansas, on September 13 and 14. According to Dr. Burton the Kansas plan has progressed more than satisfactorily and their pioneering work has been of great value to other states. Medical fees paid to Kansas physicians during the month of August were approximately $250,000 and gaining each month.

### Veterans Administration Representative To Visit Oklahoma

Arrangements have been made for Dr. Thomas Lowry, Director of the Out Patient Department of the Veterans Administration Regional Office in St. Louis, to be in Oklahoma City the latter part of September to meet with the Veterans Medical Care Committee of the Association to discuss fees to be paid for medical services rendered and the method to be selected of approving physicians who will voluntarily agree to participate in the work.

### Veterans Regional Office Established In Oklahoma City

With the establishment of a regional office of the Veterans Administration in Oklahoma City, it is anticipated that the operation of the plan will be greatly simplified inasmuch as the Executive Office of the Association will be accessible for coordination of the details.

All members of the medical profession will be contacted directly as soon as the plan is completed and it is contemplated that Councilor District meetings will be held to further explain the plan to the profession.

## OKLAHOMA EDUCATION IN HEALTH LEAGUE ORGANIZED

On September 3, 1946, at a meeting held in the offices of the Oklahoma State Medical Association, there was organized the Oklahoma Education in Health League having as its objective "to promote health education to the lay public". The following organizations had representatives attending, which constitute the present membership: American Cancer Society, Blue Cross Hospital Plan and Oklahoma Physicians Service, Oklahoma Society for Crippled Children, National Foundation for Infantile Paralysis, State Tuberculosis Association, State Dental Society; State Pharmaceutical Association, State Vocational Rehabilitation, State Department of Health, University of Oklahoma School of Medicine, Veterans Administration and the Oklahoma State Medical Association. Membership is open to organizations concerned with public health and welfare. Two-thirds vote approval for new members is required.

WILLIAM E. EASTLAND, M.D.
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The story of facilities available and the work now being done in the various fields pertaining to health matters, was the objective of the organizations participating in fair exhibition. Considerable effort was expended to make the public conscious of the public health problems and to solicit their cooperation in furthering the improvement of the situation.

The first activity of the League was the "Life Show" presented through the Oklahoma State Fair of 1946. It is anticipated that this is only the first effort to carry out the objective of the organization and that there will be more frequent and larger health educational activities. Considering the broad field of health education, without doubt the opportunities for service by the new league will be innumerable.

### V. A. Hospital Construction Program Changes In Oklahoma

The Naval Hospital buildings at Norman have been transferred to V. A. for temporary use as a 750 bed N. P. hospital. A permanent 750 bed N. P. hospital for treatment of cronic cases will be constructed at the Norman site.

A 1000 bed general medical and surgical hospital will be constructed at Oklahoma City, with about 50 beds to be set aside for treatment of acute N. P. cases. This project is subject to a future appropriation.

The Central Oklahoma State Hospital for mental patients being in Norman, and the University of Oklahoma Medical School being in Oklahoma City, make both sites logical locations for V. A. hospitals.

## SURPLUS PROPERTY SITUATION STILL CONFUSING

Through recent correspondence with the War Assets Administration the information has been developed that the program is not so constituted as to give service to the veteran when he is in need of surplus property adaptable to his practice.

It can be seen from the following letter from the W. A. A. that the physician veteran must wait his turn and depend upon being advised at some future date.

"Reference is made to your letter of August 26 regarding availability of 'set aside' items.

"We regret to advise you we do not have any dental or medical 'set aside' items available on our inventory at this time. However, if and when this equipment is available the veterans who are certified will be notified and given an opportunity to purchase.

"If we can be of further assistance to you in this matter kindly advise this office."

### RED CROSS REQUESTS DOCTORS TO RETURN OUTDATED PLASMA

Blood plasma which becomes out-dated should not be destroyed, as there are several uses to which it can be put, according to word received at the State Department of Health from Dr. Raymond F. Barnes, director of medical and health services, American Red Cross. Physicians having outdated plasma on hand are requested to send it to the American Red Cross, Midwestern Area Storeroom, 1709 Washington Avenue, St. Louis, Mo.

It has also been noted that a number of physicians have been filling out the report forms found in the boxes of plasma. This is not necessary, Dr. Barnes pointed out, as the army is no longer interested in the questionnaire.

### Governor Appoints Committee on Standardization

Governor Robert S. Kerr has made the following appointments to the Committee on Standardization:

Earl McBride, M.D., Oklahoma City, was reappointed to serve as Chairman.

I. F. Stephenson, M.D., Alva, Vice Chairman.

Mr. Joe N. Hamilton, Oklahoma City, appointed Secretary.

Committee members appointed as follows: J. F. Park, M.D., McAlester; E. Eugene Rice, M.D., Shawnee; Floyd Newman, M.D., Shattuck; and M. M. Williams, D.D.S., Chickasha.

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# Have You Heard That...

At the Annual Meeting of the American College of Chest Physicians held at San Francisco June 27-30, 1946 *Dr. Robert M. Shepard*, Tulsa, was re-elected Governor of the College for the State of Oklahoma.

*Dr. Ben Bell*, having served over three years in the Navy as a Neuro-psychiatrist, has recently returned to the Coyne Campbell Sanitarium.

*Dr. G. F. Mathews*, Commissioner of Health, announced the appointment of Dr. S. S. Kirkland of Sallisaw, as County Health Superintendent of Sequoyah County, effective September 6, 1946.

*Dr. Harvey Roys* of Norman, recently released from the service, has entered into practice with his brother in Seattle, Washington.

*Drs. Thomas H. Briggs and Joseph S. Fulton* of Atoka recently attended a meeting of the Chamber of Commerce of Coalgate concerning the construction of a hospital in that City.

*Dr. C. E. Northcutt* of Ponca City, member of the Council, is now back to his practice after having been hospitalized by an automobile accident.

*Dr. Vance F. Morgan* has announced the opening of his office at 113 W. Oklahoma Avenue in Guthrie. Dr. Morgan is an O. U. graduate and in addition to serving an internship at University Hospital in Oklahoma City has completed post graduate work at Johns Hopkins and Vanderbilt Universities.

*Dr. Howard F. Turner*, Tulsa, has been named medical director for the American Airlines in Tulsa.

A former Picher physician, *Dr. Calmes P. Bishop*, is the new director of the Muskogee city-county health unit.

A corporation has been formed in McAlester to build a new Medical Arts Building. *Dr. T. H. McCarley* is president; *Dr. F. T. Bartheld*, Vice-President; and *Dr. E. D. Greenberger*, Secretary-Treasurer.

*Dr. Ralph McGill*, Tulsa, has returned to his home in the Mayo Hotel after quite a long siege in the hospital.

It looked like a conclave of old grads when recently *Dr. James Stevenson*, Tulsa. *Dr. V. C. Tisdal*, Elk City, and *Dr. Charlie Rountree*, Oklahoma City, arrived simultaneously in the Executive Office, each without knowing the others were present.

Latimer County has welcomed *Dr. G. R. Booth, Jr.*, who has recently opened his office in the Donathan Building in Wilburton, following his release from the armed forces.

*Dr. James Hood*, Norman, has been appointed as Director of Student Health Service at the University, having succeeded *Dr. John Y. Battenfield* who will go to Liberia in the employment of Firestone Tire and Rubber Co.

*Dr. Edward Thorp*, a graduate of the University of Oklahoma in 1942, has opened offices in Cushing.

*Dr. Morris Smith* has resumed his practice in Guymon after a number of months rest. He has entered into partnership with *Dr. E. L. Buford*, who had had his office in the Smith Clinic Building for several months.

The appointment of *Dr. P. H. Anderson* of Anadarko, effective September 15, 1946, as County Superintendent of Health of Caddo County, has been made by Dr. Grady F. Mathews, Commissioner of Health.

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PRESCRIBED BY MANY DOCTORS ... You also may want to utilize Daricraft as a solution to your infant feeding problems, as well as in special diets for convalescents.

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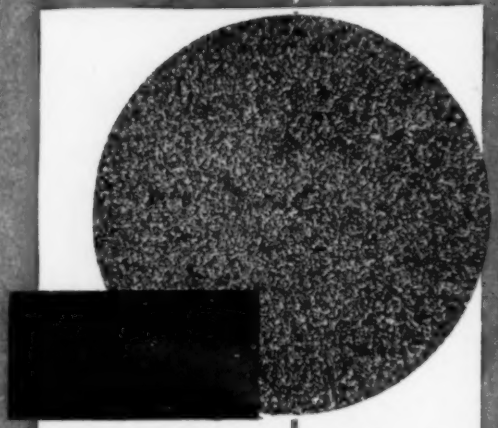
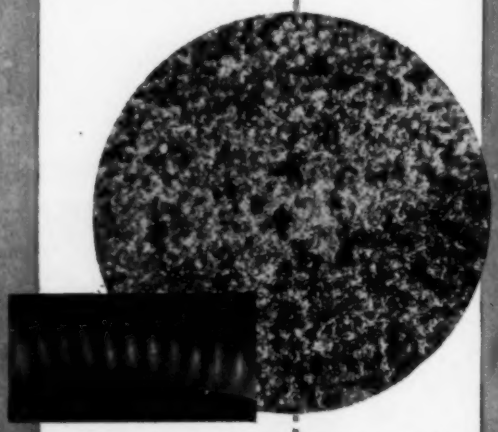
# Through the MICROSCOPE



No. 1 Unretouched photomicrograph of the dome (enlarged 10 diameters) and the rim (inset) of a "RAMSES" Flexible Cushioned Diaphragm.



No. 2 Unretouched photomicrograph of the dome (enlarged 10 diameters) and the rim (inset) of a conventional-type diaphragm.

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The discerning eye of the microscope reveals notable advantages of the "RAMSES"* Flexible Cushioned Diaphragm.

Only the "RAMSES" has the patented rim construction which provides both a wide, unindented area of contact with the vaginal walls, and a cushion of soft rubber to buffer spring pressure.

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# Hits, Runs, Errors

AUGUST 21: The highschool All Stars are in town and these boys are certainly a credit to the many cities from which they come. Give them a hand when they get home. Bill Harkey, attorney for the Medical Boards, called and there is a conference over the Optical Store situation. Seems they are only now operating in Okmulgee and Ada, also looks as if the Attorney General is going to take action. Haskell Newman, M.D., in from Shattuck, but not flying his airplane. Who said county practice can't be wonderful? Dr. Coyle from Chandler, we forgot his first name, wanted to know about locations. He is just out of the army. We gave him the list and he will be back. The program for the County Medical Societies Officers' Conference is about complete and with pardonable pride may we say it looks like the best we have ever had.

AUGUST 22: Word from Dr. Day that Dr. Clulow passed away in Tulsa. We will try to get some help for the hospital. Lunch and afternoon conference with Attorney for London and Lancashire who carry our Malpractice Insurance. More correspondance and telephone conversations about On-the-Job training. Now we are discussing interns and residents.

AUGUST 23: Frankly, this was a lazy day. The Journal did get some attention and we are now going to analyze the advertising for the past five years and see if we can arrive on a formula for the number of pages to be printed in ratio to pages of ads. Try this sometime; its fun. Fondren inherits this little problem. North All-Stars beat the South 32-6 and I watched the game on a free ticket from Dr. C. R. Rountree and family.

AUGUST 24: The mail brought a letter from the A. M. A. on their radio program for 1947, the Centennial year. Each state will have its history of medicine dramatized. A note from the President finds Dr. Kuyrkendall in Minneapolis and we are now looking for a box of iced fish. Dr. Northcutt of Ponca City writes that he is on the mend following an accident and invites us to Ponca City on the 12th of September. We are going.

AUGUST 6: First returns received on survey concerning Special Train to Southern Medical Meeting. Looks good. Fondren has a meeting with the groups who will exhibit at the State Fair and things are moving along. William Brooksher, M.D., Secretary of Arkansas Society called concerning Veterans Fee Schedule and suggested Oklahoma and Arkansas try and work together. Of course, this is agreeable.

AUGUST 27: To Rotary for lunch with Dr. John Lamb and President Cross of the University of Oklahoma and a visit about the many new plans for the Medical School. A great day is dawning and everyone must help. A letter from a secretary of a chamber of commerce concerning building a hospital. It seems, according to the letter, that the doctors in the town have to take all of their "obesity" cases to another town for delivery. Something new in medicine.

AUGUST 28: Our genial Secretary, Dick, is on "tour" (???) today — out in West Indian Territory, so pinch-hitter Fondren is the "batter-up". An early caller of the morning was Dr. Brooks of Phillips, Texas. The doctor was in town on business with the Medical School, but also gave us a "hello". The mail brought in increasing data from the various organizations participating in the Health Show of the coming State Fair. Big meeting for all exhibitors scheduled for next Tuesday night, September 3, at which time final arrangements on this Fair Business will be culminated. All signs indicate that some very interesting displays are to be presented at the Fair this year. By the time this is in print, perhaps you will have seen the exhibits — please let us have your comments. Constructive criticism will help us to do a better job next year.

AUGUST 29: Fondren swinging at the ball again today — Dick did return to the office this morning — reported considerable rain on his round trip to western Oklahoma. Luncheon with Dr. John Burton and round tab'e discussion of the Veterans Care problem. Secretaries planned trip to Tulsa tomorrow to start surveying the Annual Meeting situation.

AUGUST 30: Off to Tulsa at 7:00 A.M. First stop — Mayo Hotel to see Manager, Mr. Bentley about the Annual Meeting. From there to Jack Spear's Tulsa County Medical Society Office. Dr. James Stevenson graciously invited us to lunch following which we attended latter part of Tulsa County Medical Society's Board of Trustee meeting. Then to Chamber of Commerce Convention Chairman's and Mr. Bethel at Blue Cross for an interesting and instructive tour of Blue Cross Headquarters.

AUGUST 31: Nothing of interesting news value this Saturday A.M.

SEPTEMBER 2: Since each organization and person consulted planned no operations, the Association's office did not open this Labor Day.

SEPTEMBER 3: Paul B. Champlin, M.D., James Stevenson, M.D., F. Redding Hood, M.D., McClain Rogers, M.D. and Dick Graham departed last evening for St. Louis to attend National Physicians Committee meeting. Final Fair meeting in Library at 8:00 P.M.

SEPTEMBER 4: State Fair meeting last night, 13 participating organizations represented. "Oklahoma Education in Health League" created. Crippled Children's Committee meeting scheduled for today postponed — not enough members could attend.

SEPTEMBER 5: Dick Graham back from trip to St. Louis. Discussion with Dr. John Burton on coming Veterans Care Committee meeting. Had a visit from Dr. Talley concerning a location for a physician.

SEPTEMBER 6: Telephone conference with Harry Kornbaum concerning Special Train to Southern Medical. Tried to talk to Warren Humphreys of Erwin-Wassey on advertising program. Too many people still

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taking vacations. Exhibit program at State Fair causing trouble — too many people with different ideas.

SEPTEMBER 7: Telephone conversation with Dr. Collier on Venereal Disease problem. Talked to Dr. Newman concerning Woodward County Medical Society meeting scheduled for September 11. Muriel Waller called in regard to the Association's Special Assessment. Dr. Coyle in the office to find out about insurance and procedure for joining the Association after he establishes his new location.

SEPTEMBER 8: To Shawnee for a meeting of the Executive Committee of the Seventh Councilor District. Clinton Gallaher, M.D., Councilor, and Mrs. Clint had a wonderful buffet for those in attendance. All but two counties represented, and we were glad to again visit with Dr. Rollins of Prague, Dr. Felts of Seminole, Dr. Robberson of Wynnewood, Dr. Hicks of Holdenville, Dr. Morey of Ada and Dr. Newlin of Shawnee.

SEPTEMBER 9: Dr. Hackler in office this A.M. to help iron out a few more Fair problems and discuss publicity angle. Dr. Rountree dropped in to catch up on current matters — the doctor just returned from Colorado vacation. Harry Kornbaum, our travel expert, called to discuss proposed Southern Medical Meeting trip.

SEPTEMBER 10: Southern Medical Trip plans discussed at great length with Harry Kornbaum of Rainbow Travel Service. Meeting this evening with Harry and Dr. Carroll Pounders. Trip promises to be a great one.

SEPTEMBER 11: Dean Gray, Dr. Lamb, Dick and I went out to Shattuck to attend the Woodward County Medical Society meeting. State Commissioner of Health, Dr. Grady Mathews, was also there to comment on Hospital Construction Act. Dean Gray found his first trip to Western Oklahoma very interesting.

SEPTEMBER 12: Late arrival this A.M. — up late last night because of return trip from Shattuck. Many telephone conversations with organizations participating in Fair program. Dick making arrangements to go to Topeka tomorrow in connection with Veterans Care Program.

SEPTEMBER 13: Dr. Hewitt from Muskogee in office early to discuss available opportunties for returning veteran doctors — gave him list made from survey before his departure. Trying desperately to get articles for Journal lined up; already two days past deadline. Oklahoma Executive Council luncheon at noon — must see the printer this afternoon and work on Journal mailing list problem which is always with us.

SEPTEMBER 14: Dr. Ellis in office to recheck location list. Dr. Stevenson called from Tulsa to arrange meeting of his Medical Education and Hospital Committee. Still trying desperately to get Journal articles assembled for October issue. Working on Dr. McBride's Committee meeting, scheduled for tomorrow morning.

SEPTEMBER 15: Dr. Earl D. McBride held meeting of the Crippled Children's Committee this morning in the Executive Office to discuss fee question for polio patients.

SEPTEMBER 16: Fair Exhibit from A. M. A. arrived this morning. Dick went to McAlester to see Dr. Kuyrkendall and on to Coalgate for hospital meeting.

SEPTEMBER 17: Dr. Glissman paid us a visti this A.M. Dick back from McAlester and Coalgate. Secretaries Conference to be September 29th.

SEPTEMBER 18: Conference with Advertising Agency concerning ads this A.M. Visited the Cancer Society headquarters to see their program for the big meeting the 27th.

SEPTEMBER 19: A busy day. To Chickasha for a 9:00 o'clock meeting with the Chamber of Commerce, and back to the office for a meeting of Dr. Stevenson's Medical Education and Hospital meeting and at 2:30 to Ponca City with Dr. Lingenfelter and Dean Gray. At Ponca City we saw the joining together of Kay and Noble County Medical Societies and a steak dinner to boot. Haskell Smith of Stillwater says this column is well named as our "errors" are more than our hits. We admit we don't know the name of the damn at Cody, Wyoming.

SEPTEMBER 20: This is the day before the Fair and everyone is busy preparing booths for the gala opening tomorrow.


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INJECT 1 to 3 cc. Metrazol as a restorative in circulatory and respiratory failure, in barbiturate or morphine poisoning and in asphyxia. PRESCRIBE 1 or 2 Metrazol tablets for a stimulating-tonic effect to supplement symptomatic treatment of chronic cardiac disease and fatigue states.

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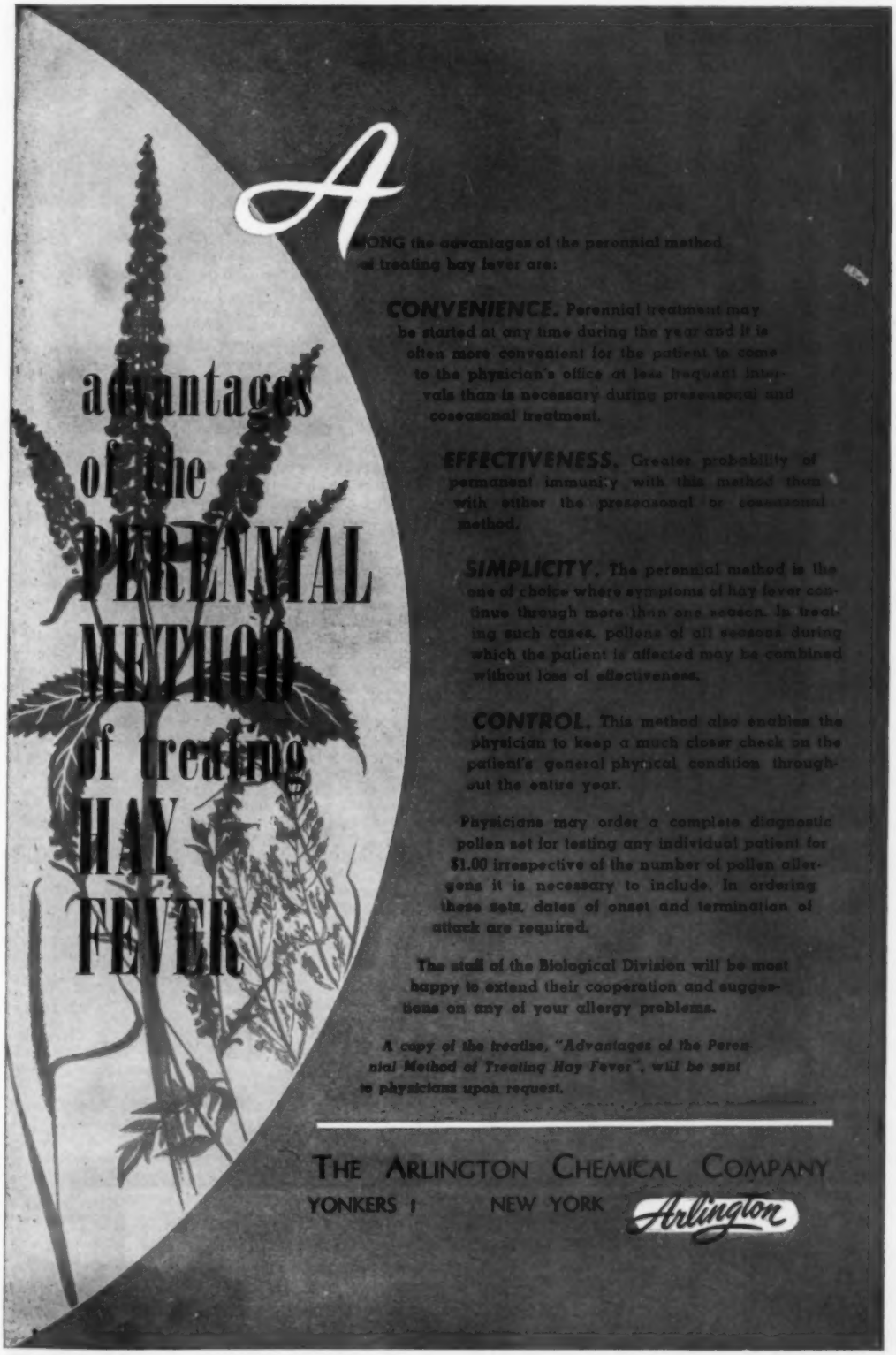


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MONG the advantages of the perennial method of treating hay fever are:

**CONVENIENCE.** Perennial treatment may be started at any time during the year and it is often more convenient for the patient to come to the physician's office at less frequent intervals than is necessary during preseasonal and coseasonal treatment.

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The staff of the Biological Division will be most happy to extend their cooperation and suggestions on any of your allergy problems.

*A copy of the treatise, "Advantages of the Perennial Method of Treating Hay Fever", will be sent to physicians upon request.*

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# Book Reviews

*PERIPHERAL VASCULAR DISEASES.* Edgard V. Allen, B.S., M.A., M.D., M.S. in medicine, F.A.C.P. Division of Medicine, Mayo Clinic, Associate Professor of Medicine, Mayo Foundation, Graduate School, University of Minnesota; Diplomate of the American Board of Internal Medicine; Nelson W. Barker, B.A., M.D., M.S. in Medicine, Mayo Clinic, Associate Professor of Medicine, Mayo Foundation, Graduate School, University of Minnesota, Diplomate of the American Board of Internal Medicine; Edgar A. Hines, Jr., M.D., B.S., M.A., M.S. in Medicine, F.A.C.P., Division of Medicine, Mayo Clinic, Associate Professor of Medicine, Mayo Foundation, Graduate School, University of Minnesota. First Edition, 386 illustrations, eleven colaborators, 871 pages. W. B. Saunders Company, Philadelphia, Pa.

This is an entirely new book. The authors are all competent men, having had extensive experience in the field of peripheral vascular diseases. Eleven of the authors' colleagues have contributed to the book, which is dedicated to Dr. George Elgie Brown, who is credited with doing much of the original work and inspiring these authors to write this book.

A discussion of hypertension and vascular diseases of certain viscera have been omitted. There is no attempt to cover the technical aspects of sympathetic nervous system surgery.

Other than the above omissions, the field of peripheral vascular diseases, including disorders of lymph vessels and veins, is well covered in a clear, concise manner. The authors have expressed their own opinions freely on many controversial matters, and have done an excellent job of avoiding cumbersome repetition, and have wisely omitted burdening their book with researches which do not have a direct clinical bearing.

The book attempts to clarify much of the confusing terminology by devoting the first chapter to a definition of terms.

In reading through this book, one is particularly struck by the open-minded manner, and complete lack of prejudice, with which the authors approach the subject. Simplicity with a thorough understanding and reliance on basic physiologic principles, both as regards diagnosis and treatment, is shown throughout the book. The authors do not rely on any single method or type of treatment (such as alternating suction and pressure, intermittent venous occlusion, contrast baths, sympathetic surgery, intravenous typhoid, etc.) in the care of occlusive vascular disease. Rather, they rely on a combination of several methods of treatment as illustrated by the outline for handling thrombo-angitis obliterans. (Pages 469-70).

I believe that this book is a very worthwhile contribution to a widely neglected field of medicine and that it will go far toward bringing an unanimity of opinion in the proper care of patients afflicted with peripheral vascular diseases.—Turner Bynum, M.D.

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*GRACIAN'S MANUAL.* Martin Fischer, a doctor and professor in the University of Cincinnati. Second and revised edition. 300 pages. Charles C. Thomas, Springfield, Illinois.

"Gracian wings across some fifty odd years of history like the flight of a Mother Carey's chicken. Out of a gray obscurity, he flashes into the visible sky—black, swift, unafraid, screaming his song," Gracian lived in the first half of the seventeenth century.

The following statement taken from the jacket of this volume stimulates the reader, and a first reading justifies the statement:

"This is not a sweetmeat for children, but a volume for men of this world—and but few of them. A new translation into English of the famous Spanish classic of Padre Balbasar Gracian. A practical manual of self-instruction, absolutely unique, and peculiarly appropriate to the thinking man of these perilous times."

Each paragraph is marked by scarlet numerals. While the "art of worldly wisdom" appears on every page, this is not a book to be read at a single sitting, but to be placed at the bedside or in the traveling bag for periodic perusal in small doses during periods when the time is propitious. It would be difficult to find in any other single volume so much sound unobtrusive advice.

This book is heartily recommended to doctors who find it easy to develop the habit of thinking there is no time to read outside the field of medicine. This is a book offering genuine reward in return for spare moments.—Lewis J. Moorman, M.D.

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*MODERN MANAGEMENT IN CLINICAL MEDICINE.* Frederick K. Albrecht, M.D., S. A. Surgeon, ....U. S. Public Health Service Kansas State Tuberculosis Consultant; Formerly Clinical Director, U. S. Marine Hospital, Baltimore, Maryland. 1,238 pages. 1946. The Williams and Wilkins Company. $10.00.

This book was obviously conceived by the author as a volume intended for the doctor's office. A tremendous amount of material has been compiled with the objective of bringing to the medical practitioner a readily available wealth of known facts, as well as new facts. It has been accomplished in such a clever way that the reader need consume only a minimum of time in obtaining necessary information on a particular subject. Excellent photographs and x-ray plates representing disease entities are presented throughout the book.

For those who wish to do more reading on any particular subject or desire more complete information, Dr. Albrecht has included at the end of each chapter, a bibliography of the latest references on each subject.

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The most recent developments and discoveries in diagnostic procedures, special techniques and therapy are presented in a readily available form. Such procedures as hepatosplenography, peritencoscopy, and electroencephalography are discussed. There are excellent chapters on vitamin deficiencies, tropical diseases of postwar importance, chemotherapy and geriatrics. Therapy in all subjects, not only includes recent discoveries, but the common trade names of drugs and pharmaceutical preparations are included which should eliminate difficulty in obtaining and prescribing the proper drugs.

The Appendix includes illustrations and descriptions of procedures such as thoracentesis, sternal bone marrow cavity of administration of fluid and tidal bladder drainage apparatus. The desire of the author to make this book a complete reference for the busy physician is further manifest by his brief but concise description of the preparation of counter irritant such as poultices, hot and cold packs and even the placing of a patient in a wheel chair is not omitted.

The last 11 pages of the Appendix are devoted to the various types of diet, completely described in outline form. Reduction diets, high caloric diets and those of Karrell and Muelengracht are only a few of those included.—George S. Bozalis, M.D.

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*NEW AND NONOFFICIAL REMEDIES.* 1946 edition. 770 pages. 24 chapters. Council on Pharmacy and Chemistry, American Medical Association.

"This book is published by the Council on Pharmacy and Chemistry which is a standing committee appointed by the Board of Trustees of the American Medical Association, to consider medical preparations offered by pharmaceutical manufacturers for prophylactic and therapeutic use by the physicians." The Council is composed of eighteen members and forty consultants, whose hallmark of approval should be unquestioned. This publication not only lists and describes the product, but also (which is quite essential) lists the manufacturers which put out the product. Manufacturers put out so many specifics that only cure for two or three months and detail men with highpressure salesmanship flood the market with these nostrums that are either inert or possibly toxic (Messengil sulph solution an example). Research facilities should preceed all new products and hence the desirability of this to me, to see if the stamp of approval has to be given by this group of financially disinterested and scientifically interested scientists recommend it. There is a voluminous bibliography on products not acceptable, and show the source of information about such articles.

The Oklahoma University School of Medicine furnishes each graduate with one of these valuable and up-to-date books for perusal. Each physician should have one at his elbow for a check up on the claims of products made more for financial gain and beating their competitors to it, than for the humanitarian aspect. These acceptable products are approved for only three years, and if unwarranted claims of efficiency or change in the formulary, thereby it is a just cause for its being dropped. Since the metric system has displaced the apothecaries weights and measures, there is a chapter and table of equivalents for those not used to the new system.

Addend: Revision of the Rules of the Council on Pharmacy and Chemistry, J.A.M.A., 131-215-219, May 18, 1946, curtails the firms from exploiting remedies for profit, using a few better articles for "window dressing" than for serious promotion. The Council aims to insist that the major business of a firm should be in acceptable articles before any of its products would be accepted by the Council. This, of course, is aimed for the protection of the physician as well as the buying public. —Lea A. Riely, M.D.


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# Medical Abstracts

**ATOPIC CATARACTS. Frederick C. Cordes and Rafael Cordero-Moreno. American Journal of Ophthalmology. Vol. 29, pp. 402-407. April, 1946.**

The association of skin diseases with cataract formation is a generally recognized fact. Cataract may develop in the course of neurodermatitis, poikiloderma atrophicans vasculare, and scleroderma. Cataracts have been also associated with keratosis follicularis, telangiectasis, myxedema, and certain abnormities of the hair (alopecia, aplasia pilaris, etc.)

The dry form of neurodermatitis has been also designated as atopic dermatitis. Atopy is a form of allergy, and includes hayfever, asthma and eczema. It occurs in the presence of specific reagins in the blood stream. These reagins enter the body during embryonic life, and the sensitivity thus transferred will remain until about the middle life. During infancy it is manifested on the skin by eczematous lesions of the weeping, crusty type. In childhood, papular eruption is present, which may or may not be accompanied by asthma. In adult life, the lesions consist of elevated papules and dry, scaly, lichenified plaques. The course of the disease is chronic.

Cataract formation as part of the picture of neurodermatitis or atopic dermatitis may begin at any time. A typical atopic cataract is a white milky plaque in the pupillary area, localized either on the anterior or on the posterior pole of the lens, involving the superficial layers of the lens cortex. The early changes are granular deposits in the anterior capsule, and an increase in the prominence of the Y suture lines. The mature cataract appears gray or light cream in color. Striation of the lens fibers is often present as are small punctate opacities and iridescent crystals. Later, these small opacities will coalesce.

The incidence of cataract in atopic patients is not determined. Some authors mention 10 per cent, which is a rather high rate. The cataract is apt to appear in puberty and young adult life. Sex has no apparent influence in the incidence of atopic cataract.

The mechanism of the formation of the lens opacities is not certain. It may be that lens opacities are but manifestations of an ectodermal disease. It is possible that in the course of allergy the lens may become a so-called shock organ, i.e., an organ which is specially sensitive against the reagins of the blood.

The treatment of such cases is surgical. Most of the patients fall in an age group wherein intracapsular extraction is not recommended since there is a tendency in the capsule to rupture.

The authors report four cases: all of them showed atopic dermatitis with cataract formation. One patient was a 15-year old boy whose father had had asthma for many years and one brother had hayfever; he himself suffered from asthma and eczema; his cataract was bilateral subcapsular. A 17-year old girl had a type of skin disease which was a mild eczema of the face; her cataract was also bilateral, and was complicated by retinal detachment.—M.D.H., M.D.

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**SOME CONTRIBUTIONS OF ENDOCRINOLOGY TO OBSTETRICS AND GYNECOLOGY. E. C. Hamblen, M.D., American Journal of Obstetrics and Gynecology, Vol. 51, No. 6, pp. 796-803. June, 1946.**

Dr. Hamblen briefly reviews endocrinology, with especial reference to the ovary, from the pioneer work of Allen, Loeb, and others to the present time. He then discusses various technical phases of present day conceptions. He states that the climateric patient is too often the victim of very poor therapy. Quoting from his article " . . . In the climateric woman estrogen therapy has often been too frequent and too uncritical . . . ". "Few climateric women require estrogens . . . ". " . . . Injectional therapy is not necessary—its psychologic approach is unhealthy . . . ". "Infrequent injections of large amounts (of estrogen) are an ideal formula for

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keeping the entire endocrine system in a state of flux and chaos. . . .'' ''If estrogens are necessary, the daily dose should not be larger than .5 mg. of Stilbestrol, or its equivalent.''

To the above might be added that estrogens are potentially dangerous to women who have certain breast tumors, or women who have a family history of cancer. —G.P., M.D.

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**ROENTGENOGRAPHIC INTERPRETATION OF WHAT CONSTITUTES ADEQUATE REDUCTION OF FEMORAL NECK FRACTURES: Robert T. McElvenny, Surgery, Gynecology and Obstetrics, Vol. 80, pp. 97-106; 1945.**

The author has evaluated from his own experience various ways of dealing with intracapsular fractures of the hip. Certain facts have been obtained from his study of failures, and these facts are used in formulating criteria of roentgenographic interpretation at the time of reduction and nailing of fractures. All failures have occurred in hips which have been reduced imperfectly — that is, the neck fragment has not been placed well under and well inside the head fragment. McElvenny feels that accurate placement is absolutely necessary in order that fixation may act as a guide to allow the head to settle on the neck in a position in which the weight thrust is applied directly to the fracture site, without shearing or torsion forces being exerted on the fixation material.—E.D.M., M.D.

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**TREATMENTS OF TUBERCULOSIS OF THE LARYNX WITH STREPTOMYCIN: REPORT OF CASE. Frederick A. Figi, M.D., Section on Laryngology, Oral and Plastic Surgery, H. Corwin Hinshaw, M.D., Ph.D., Division of Medicine and William H. Feldman, D.V.M., M.S., Division of Experimental Surgery and Pathology, Mayo Foundation.**

Case report of a thirty-two year old married woman, first seen at the Clinic March 16, 1945, because of hoarseness of one year's duration, progressively worse. Physical examination revealed nothing of significance, except a laryngael picture typical of papillary tuberculous laryngitis, confirmed as such by biopsy. X-ray showed some accentuation of the right hilar shadow. Acid fast bacilli were demonstrated in the suptum.

Streptomycin was administered for forty-five days, a total of thirty-four grams was given parenterally. Nebulization with streptomycin was also used. Definite improvement of the laryngeal lesion was noted by the tenth day, and the lesion had improved 75 per cent by the twenty-fifth day of treatment. After the twenty-seventh day, all subsequent biopsys, cultures, smears from the lesion were negative for acid fast bacilli. By the forty-fifth day, the lesion was practically healed. After six months there had been no recurrence, and the patient's voice remained normal.

The authors feel that streptomycin was of definite value in the treatment of this case. However, they do not necessarily anticipate the same favorable results in the usual cases of laryngeal tuberculosis which are associated with extensive, destructive, and often fulminating pulmonary tuberculosis.—H.J., M.D.

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**RAPID REPAIR OF DEFECT OF FEMUR BY MASSIVE BONE GRAFTS AFTER RESECTION FOR TUMORS. Dallas B. Phemister, Surgery, Gynecology and Obstetrics, Vol. 80, pp. 120-127, 1945.**

By means of bone grafting, amputation was avoided in two cases of locally resected bone tumors of the lower end of the femur. Two tibial grafts, each between two-fifth and one-half the circumference of the shaft and from seven to eleven inches in length, were used to bridge the defect. The grafts were part onlay and part intramedullary, with fixation by threaded wires at each end. Firm bony union was obtained in about twelve weeks in one case and in five months in the other case. At the end of three and one-half years, both grafts had been transformed into tubular bone.—E.D.M., M.D.

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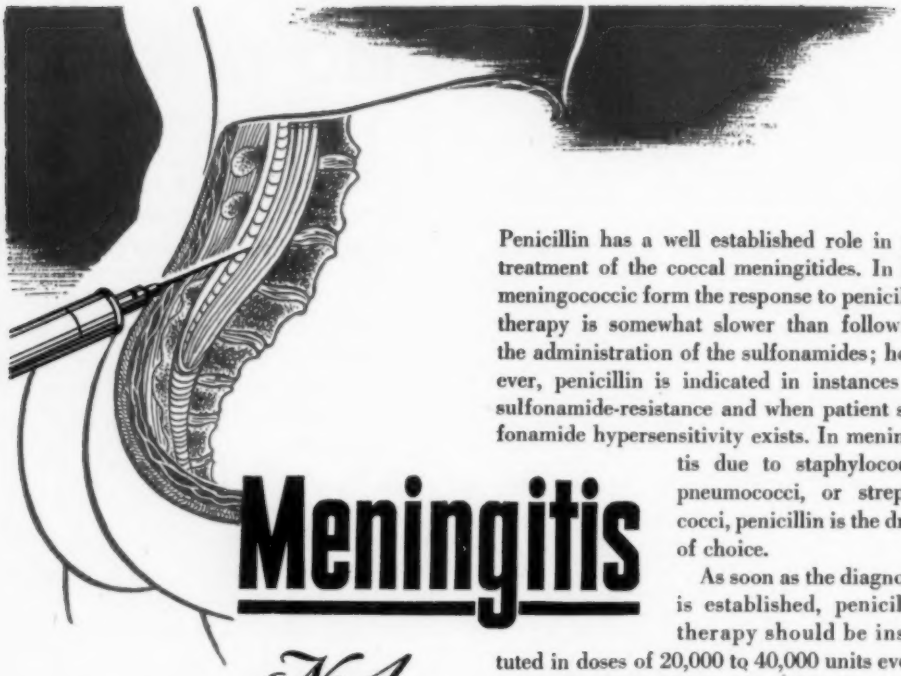

# Meningitis

*No. 4*

## IN SCHENLEY LABORATORIES CONTINUING SUMMARY OF PENICILLIN THERAPY....

Penicillin has a well established role in the treatment of the coccal meningitides. In the meningococcic form the response to penicillin therapy is somewhat slower than following the administration of the sulfonamides; however, penicillin is indicated in instances of sulfonamide-resistance and when patient sulfonamide hypersensitivity exists. In meningitis due to staphylococci, pneumococci, or streptococci, penicillin is the drug of choice.

As soon as the diagnosis is established, penicillin therapy should be instituted in doses of 20,000 to 40,000 units every two to three hours by the intramuscular route. Treatment should be thorough, and should be continued until all signs and symptoms of the infection have been absent for seven to ten days. Since penicillin administered systemically does not penetrate the subarachnoid space, intrathecal (intraspinal, intracisternal, intraventricular) administration is also required. Ten thousand units in 10 cc. of isotonic solution of sodium chloride should be injected (after withdrawal of an equal volume of fluid) once or twice daily until the spinal fluid is clear, and for four days thereafter.

When concurrent sulfonamides are indicated, they should be administered in a dosage sufficient to establish a blood level of 15 mg. per cent.

Surgical, supportive, and other measures should be employed when indicated.

---

**SPINK, W. W., and HALL, W. H.:** *Penicillin Therapy at the University of Minnesota Hospitals: 1942-1944, Ann. Int. Med. 22:510 (April) 1945.*

**WHITE, W. L.; MURPHY, F. D.; LOCKWOOD, J. S., and FLIPPIN, H. F.:** *Penicillin in the Treatment of Pneumococcal, Meningococcal, Streptococcal, and Staphylococcal Meningitis, Am. J. Med. Sc. 210:1 (July) 1945.*

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**MANDIBULAR TUMORS. A Clinical, Roentgenographic and Histopathologic Study. Louis T. Byars and Bernard G. Sarnat. Surgery, Gynecology and Obstetrics, Vol. 83, No. 3, pp. 355-364; September, 1946.**

This is a timely article and well worth reading by not only the surgeon but by the dentist and the radiologist. It reiterates a cardinal principle in the treatment of malignancy — namely, that a correct diagnosis is to only be made by microscopic study of the involved tissue.

"Because the ameloblastoma is generally considered to be a benign tumor, and because the roentgenographic pictures may be similar to malignant lesions primarily or secondarily in the jaw, it is of paramount importance to make an early correct diagnosis."

"The primary value of the roentgenogram is to demonstrate the site and extent of the multilocular lesions of the mandible."—J.F.B., M.D.

---

**ASEPTIC NECROSIS OF THE CAPITAL FEMORAL ADOLESCENT EPIPHYSEOLYSIS. Robert D. Moore. Surgery, Gynecology and Obstetrics, Vol. 80, pp. 199-204, 1945.**

Moore reviews the literature dealing with descriptions of femoral epiphyseal pathological lesions and gives a detailed account of the macroscopic and microscopic findings in two cases of slipped femoral epiphysis. The article is a purely descriptive recording and offers no explanation as to cause.

This paper may be summarized briefly, as follows: After interruption of the blood supply to the epiphysis in adolescents, the overlying articular cartilage may remain viable or may undergo partial or complete necrosis. In either case, subsequent degenerative and reparative changes apparently result in reduction of the depth of the articular cartilage, as occurs in the adult with aseptic necrosis of the head, following fracture of the neck of the femur. This is in contrast to the changes seen in Legg-Perthes disease, where rapid growth of the living articular cartilage compensates for any absorption or ossification of the cartilage which takes place after revascularization of the ossification center. Under these circumstances, the depth of the articular cartilage usually increases.—E.D.M., M.D.

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**TRAUMATIC OSTEOMYELITIS: THE USE OF SKIN GRAFTS—PART I. TECHNIC AND RESULTS. Robert P. Kelly, Louis M. Rosati, and Robert A. Murray. Annals of Surgery, Vol. 122, pp. 1-11, 1945.**

These authors report on the satisfactory use of skin grafting in the treatment of osteomyelitis. The technique is fully described. Emphasis is given to the need for a thorough preliminary saucerization and the equal need for applying the grafts so that compression can be distributed evenly to all of them. On the bases of approximately one hundred cases treated by the method described, it is believed that this is a safe form of treatment and that it will give beneficial results in the majority of instances. While occasional reports of the successful use of skin grafts in the treatment of osteomyelitis have appeared in the literature for many years, the method has not received wide use. The authors hope that this encouraging report will stimulate others to apply this treatment.—E.D.M., M.D.

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KEY TO ABSTRACTORS

M.D.H., M.D. ..... Marvin D. Henley, M.D.
G.P., M.D. ..... Grider Penick, M.D.
E.D.M., M.D. ..... Earl D. McBride, M.D.
H.J., M.D. ..... Hugh Jeter, M.D.
J.F.B., M.D. ..... John F. Burton, M.D.


# Have a Coke

When you laugh, the world laughs with you, as they say—and when you enjoy *the pause that refreshes* with ice-cold Coca-Cola, your friends enjoy it with you, too. Everybody enjoys the friendly hospitality that goes with the invitation *Have a Coke.* Those three words mean *Friend, you belong —I'm glad to be with you.* Good company is better company over a Coca-Cola.

# Obituaries

### George H. Clulow, M.D.
**1880-1946**

George H. Clulow, M.D., passed away on August 17 at the Hillcrest Memorial Hospital of Tulsa following an illness of only a few days.

Since coming to Oklahoma in 1913 Dr. Clulow had practiced in Tulsa until January 1 of this year when he accepted a position on the staff of the Western Oklahoma State Hospital at Fort Supply.

A native of Pennsylvania, Dr. Clulow was born January 15, 1880, and received an A. B. degree from Allegheny College in Meadville, Pennsylvania, in 1909 and an M. D. degree from the Western Reserve Medical School of Cleveland in 1912.

In World War I Dr. Clulow served two years with the 3rd Division and was retired with the rank of Major. He was a member of the Woodward County Medical Society, the Oklahoma State Medical Association and the American Medical Association.

Survivors include Dr. Clulow's wife, Anna, Fort Supply; a son, Mack, Tulsa, and a daughter, Doris, Tulsa.

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### T. D. Rowland, M.D.
**1870-1946**

T. D. Rowland, M.D., Shawnee physician since 1902, died in a hospital there September 3.

A native of Tippah, Mississippi, Dr. Rowland came to Shawnee after graduation from the Memphis Hospital Medical College in 1902. He was born November 3, 1870.

Dr. Rowland served overseas for one year during World War I. He was a member of the First Christian Church, the Masonic Lodge and the American Legion. He also held membership in the American Medical Association, the Oklahoma State Medical Association and the Pottawatomie County Medical Society, which he had served as president.

### E. K. Collier, M.D.
**1903-1946**

E. K. Collier, M.D., of Tipton died August 9 in a Norman hospital.

Dr. Collier was born March 25, 1903, near Elmer, Oklahoma, and was reared in Tipton, attending Marion Military University in Alabama, Georgetown University in Texas, the University of Texas, and was graduated from the University of Oklahoma with a B. S. degree and Tulane University with an M. D. degree. His practice in Tipton had extended over a period of 15 years.

Surviving Dr. Collier are his mother, Mrs. Mabel Norfleet Collier; a son, Robert John; a brother, Dr. Roy Collier, and two sisters, Mrs. Helen Anderson and Mrs. Miriam Christian.

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### Elsie Specht, M.D.
**1883-1946**

Elsie Specht, M.D., died August 20 in an Enid hospital at the age of 63.

Forty-one years of medical practice in Fairview had been completed by Dr. Specht. She was a member of the Methodist Church in Fairview, the Garfield County Medical Society, the Oklahoma State Medical Association, and the American Medical Association.

---

### L. C. Vance, M.D.
**1894-1946**

L. C. Vance, M.D., of Ponca City died September 7th of a heart attack. Services were conducted September 9th in the First Baptist Church of Ponca City.

Dr. Vance maintained offices in Ponca City from 1921 until the time of his death, during which time he had been active in many civic organizations and activities. He was a member of the Kay County Medical Society, the Oklahoma State Medical Association, and the American Medical Association.

He is survived by a daughter, Mary Ann, a son, Tex, and two sisters.


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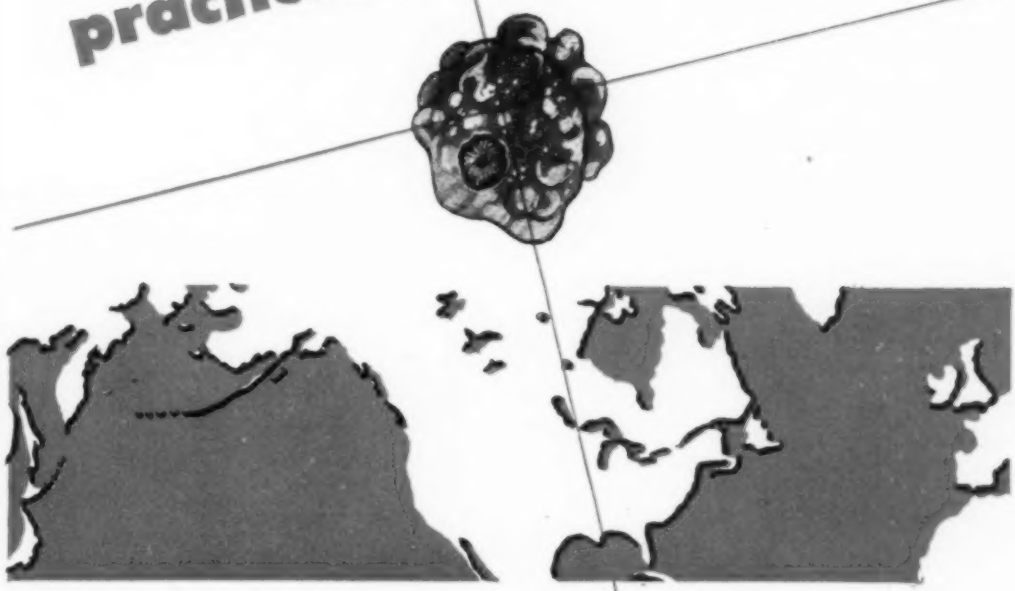

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**Laryngoscope, Feb. 1935, Vol. XLV, No. 2, 149-154.*
*Laryngoscope, Jan. 1937, Vol. XLVII, No. 1, 58-60.*

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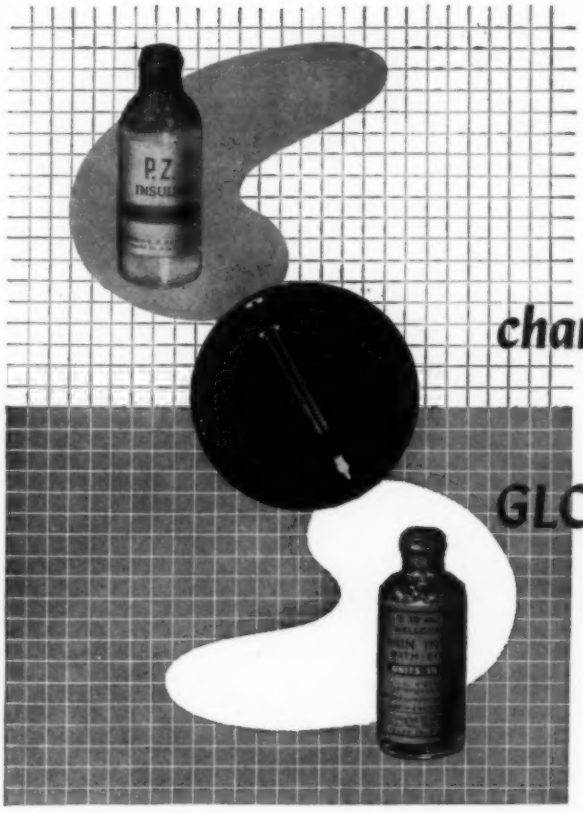


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|---|---|---|---|
| Alfalfa | W. G. Dunnington, Cherokee | L. T. Lancaster, Cherokee | Last Tues. each Second Month |
| Atoka-Coal | J. B. Clark, Coalgate | J. S. Fulton, Atoka | |
| Beckham | P. J. DeVanney, Sayre | J. E. Levick, Elk City | Second Tuesday |
| Blaine | W. F. Bohlman, Watonga | Virginia Curtin, Watonga | Third Thursday |
| Bryan | W. K. Haynie, Durant | Jonah Nichols, Durant | Second Tuesday |
| Caddo | P. H. Anderson, Anadarko | Edward T. Cook, Jr., Anadarko | Third Thursday |
| Canadian | G. L. Goodman, Yukon | W. P. Lawton, El Reno | Subject to call |
| Carter | J. Hobson Veazey, Ardmore | H. A. Higgins, Ardmore | Second Tuesday |
| Cherokee | P. H. Medearis, Tahlequah | R. K. McIntosh, Jr., Tahlequah | First Tuesday |
| Choctaw | Floyd L. Waters, Hugo | O. R. Gregg, Hugo | |
| Cleveland | James O. Hood, Norman | Phil Haddock, Norman | Thursday nights |
| Comanche | Wm. Cole, Lawton | E. P. Hathaway, Lawton | |
| Cotton | G. W. Baker, Walters | Mollie Scism, Walters | Third Friday |
| Craig | Lloyd H. McPike, Vinita | J. M. McMillan, Vinita | |
| Creek | W. P. Longmire, Sapulpa | Philip Joseph, Sapulpa | Second Tuesday |
| Custer | Ross Deputy, Clinton | C. J. Alexander, Clinton | Third Thursday |
| Garfield | Bruce Hinson, Enid | John R. Walker, Enid | Fourth Thursday |
| Garvin | M. E. Robberson, Jr., Wynnewood | John R. Callaway, Pauls Valley | Wednesday before Third Thursday |
| Grady | Roy Emanuel, Chickasha | Rebecca H. Mason, Chickasha | Third Thursday |
| Grant | I. V. Hardy, Medford | F. P. Robinson, Pond Creek | |
| Greer | J. B. Lansden, Granite | J. B. Hollis, Mangum | |
| Harmon | W. G. Husband, Hollis | R. H. Lynch, Hollis | First Wednesday |
| Haskell | Wm. S. Carson, Keota | N. K. Williams, McCurtain | |
| Hughes | Victor W. Pryor, Holdenville | L. A. S. Johnston, Holdenville | First Friday |
| Jackson | E. W. Mabry, Altus | J. P. Irby, Altus | Last Monday |
| Jefferson | F. M. Edwards, Ringling | J. A. Dillard, Waurika | Second Monday |
| Kay | L. G. Neal, Ponca City | J. C. Wagner, Ponca City | Second Thursday |
| Kingfisher | John W. Pendleton, Kingfisher | H. Violet Sturgeon, Hennessey | |
| Kiowa | Wm. Bernell, Hobart | J. Wm. Finch, Hobart | |
| LeFlore | S. D. Bevill, Poteau | Rush L. Wright, Poteau | |
| Lincoln | J. S. Rollins, Prague | Ned Burleson, Prague | First Wednesday |
| Logan | James Petty, Guthrie | J. E. Souter, Guthrie | Last Tuesday |
| Marshall | | | |
| Mayes | L. C. White, Adair | V. D. Herrington, Pryor | |
| McClain | S. C. Davis, Blanchard | W. C. McCurdy, Purcell | |
| McCurtain | J. T. Moreland, Idabel | R. H. Sherrill, Broken Bow | Fourth Tuesday |
| McIntosh | F. R. First, Checotah | W. A. Tolleson, Eufaula | First Thursday |
| Muskogee-Sequoyah | | | |
| Wagoner | R. N. Holcombe, Muskogee | William N. Weaver, Muskogee | First Tuesday |
| Noble | A. M. Evans, Perry | Jesse W. Driver, Perry | |
| Okfuskee | W. P. Jenkins, Okemah | M. L. Whitney, Okemah | Second Monday |
| Oklahoma | W. F. Keller, Okla. City | John H. Lamb, Okla. City | Fourth Tuesday |
| Okmulgee | F. S. Watson, Okmulgee | C. E. Smith, Henryetta | Second Monday |
| Osage | E. C. Keyes, Hominy | Vincent Mazzarella, Hominy | Third Monday |
| Ottawa | C. F. Walker, Grove | W. Jackson Sayles, Miami | Second Thursday |
| Pawnee | H. B. Spaulding, Ralston | R. L. Browning, Pawnee | |
| Payne | F. Keith Oehlschlager, Yale | C. W. Moore, Stillwater | Third Thursday |
| Pittsburg | Millard L. Henry, McAlester | Edward D. Greenberger, McAlester | Third Friday |
| Pontotoc-Murray | John Morey, Ada | R. H. Mayes, Ada | First Wednesday |
| Pottawatomie | F. P. Newlin, Shawnee | Clinton Gallaher, Shawnee | First and Third Saturday |
| Pushmataha | John S. Lawson, Clayton | B. M. Huckabay, Antlers | |
| Rogers | W. A. Howard, Chelsea | P. S. Anderson, Claremore | Third Wednesday |
| Seminole | Clifton Felts, Seminole | Mack I. Shanholz, Wewoka | Third Wednesday |
| Stephens | Everett King, Duncan | Fred L. Patterson, Duncan | |
| Texas | Daniel S. Lee, Guymon | E. L. Buford, Guymon | |
| Tillman | H. A. Calvert, Frederick | O. G. Bacon, Frederick | |
| Tulsa | John C. Perry, Tulsa | John E. McDonald, Tulsa | Second and Fourth Monday |
| Washington-Nowata | Ralph W. Rucker, Bartlesville | L. B. Word, Bartlesville | Second Wednesday |
| Washita | L. G. Livingston, Cordell | Roy W. Anderson, Cordell | |
| Woods | John F. Simon, Alva | O. E. Templin, Alva | Last Tuesday Odd Months |
| Woodward | T. C. Leachman, Woodward | C. W. Tedrowe, Woodward | Second Thursday |